LIBERAL DIETS IN THE TREATMENT OF DIABETES MELLITUS

by

Constance Catherine Forsyth,
M.B., Ch.B.

A Thesis submitted to the Faculty of Medicine of the University of Edinburgh for the degree of Doctor of Medicine.

June 1949.

LIST OF CONTENTS.

LIBERAL DIETS IN DIABETES MELLITUS.

Chapter I.

INTRODUCTION.

Before the discovery of insulin, strict dieting was the only method of treating diabetes mellitus; the aim of this treatment was to obtain sugar-free urine and normoglycaemia. Such a method reduced the incidence of ketosis and prolonged the lives of diabetics but it necessitated severe undernutrition and many patients were consequently very thin and tired. Mild obese diabetics, on the other hand, did very well, and restricted diets are still universally used for such patients.

The discovery of insulin revolutionised the prognosis for the non-obese adult diabetic and diabetic children. Mere existence was transformed into abundant life.

In the years following the discovery of insulin, the necessity for undernutrition has come to be questioned and also the ideal of normoglycaemia.

More liberal forms of dietetic treatment enable patients to live freer lives with great psychological benefit.

The present investigation covers a period from October 1945 to March 1949. It entails a study of a very liberal method, in which patients are given diets entirely free except for the avoidance of raw sugar, jam and sweets. Insulin is used to control symptoms. Patients are fully nourished and no attempt is made to maintain normal blood sugar levels.

The short-term results are given in the following pages, along with a brief discussion of the relevant ideas from the vast literature on Diabetes Mellitus.

Chapter II.

HISTORICAL BACKGROUND.

An abnormal polyuria, now believed to have been diabetes, was described in the Papyrus Ebers written in Egypt about 1500 B.C.

The Roman, Aulus Cornelius Celsus (30 B.C. - 50 A.D.), left a good description of the disease and it was named dia-betes, which means "to pass through", by Aretaeus, a Greek Physician living in Alexandria in the second century A.D. He described it aptly as "a liquefaction of the flesh and bones into urine" but made no reference to the presence of glycosuria.

Nevertheless, this fact was noted by Chinese and Japanese writers in the third century who found that dogs were attracted to the sweet urine.

Hindu medical writings of the sixth century refer to diabetes as Madhumeha, or honey urine.

Avicenna (980-1027 A.D.), an Arab doctor, also recorded that the urine left a residue like honey.

The name diabetes mellitus, as distinct from diabetes insipidus, was established in the seventeenth century, by Thomas Willis of Oxford University. Willis is probably better known for his description of the Circulus Arteriosus at the base of the brain.

The treatment of diabetes was begun empirically by John Rollo, an Englishman, who found that a diet of animal foods and the avoidance of starches improved diabetic patients.

Bouchardat (1806-1886) emphasised the importance of undernutrition. He observed the disappearance of glycosuria in diabetics when food was scarce during the siege of Paris. He instituted fast days - "manger le moins possible" - and advocated fats and alcohol as a source of energy.

In 1903, Naunyn gave several maxims for the treatment of diabetes, some of which are given below.

> "When the diabetic is free from sugar, his tolerance usually increases. Therefore aim to render the patient sugar free and keep him aglycosuric."
>
> "Limitation of the total diet with resulting disencumbrance of the entire metabolism brings about a favourable result."
>
> "Reduction of carbohydrates and of proteins for the removal of glycosuria."
>
> "Sugar production from fat does not play such an important rôle as to influence diabetic glycosuria to any notable extent. For this reason and on account of its high calorie value, fat is the most valuable food substance for the diabetic."

Von Noorden in Germany, Guelpa in France and Woodyatt in America advocated subcaloric diets early in the twentieth century, but it was Allen's classical experimental work which placed the Theory of Undernutrition on a rational basis.

He taught that diabetes mellitus was a disease of total metabolism and not of carbohydrate metabolism alone. His treatment consisted of starvation days and diets of low total calorie value in which fat was cut along with protein and carbohydrate. Joslin also advocated relatively low fat intakes to prevent ketosis. Reduction of weight and freedom from glycosuria were the fundamental aims in treatment.

Some writers believed that high fat diets were well tolerated and used fat as the main source of calories for diabetic patients. Petrén (1922) and Newburgh and Marsh (1920) advocated high fat-low carbohydrate diets containing six times as many grammes of fat as carbohydrate.

Before the discovery of insulin, therefore, the treatment of diabetes mellitus was dietetic. The Undernutrition Principle was widely accepted and low calorie diets were prescribed. To a degree which varied from clinic to clinic, fat was the major source of calories.

Chapter III.

THE THEORY OF UNDERNUTRITION.

For empirical reasons, the idea of undernutrition treatment for diabetes mellitus had been gaining ground for several years before being placed on a scientific basis by the experimental work of Allen (1914, 1917, 1923).

He found that dogs made diabetic by the removal of nine-tenths of the pancreas could be kept alive on restricted diets consisting of protein and fat, but died if allowed full diets containing starches.

Dogs with seven-eighths or five-sixths of the pancreas removed, which were not diabetic after the operation, could be made so by excessive feeding. When they were changed over to a strict diet early, the signs of diabetes disappeared, but even fasting failed to cure the glycosuria in dogs who had received high diets for some time.

Loss of weight from dietary restriction alleviated the dogs' diabetes. However, the effect was not due purely to the loss of weight as the amputation of a limb did not produce the same result.

That total calorie restriction was the important factor, rather than carbohydrate reduction alone, was

shown by the failure of normal calorie diets of low carbohydrate-high fat type to lessen the severity of the diabetic condition.

Allen concluded that diabetes mellitus was a disease of total metabolism and not of carbohydrate metabolism alone and that an otherwise insufficient amount of "ambo-ceptor" may become sufficient when the metabolism of the body is artificially diminished.

In the application of his experimental findings to human subjects he prescribed subcaloric diets in which carbohydrate and fat were reduced.

The emphasis on undernutrition as of prime importance was in contrast to dietary schemes which cut carbohydrate and protein and substituted high fat contents which were thought not to produce blood sugar. The fact that these diets were subcaloric was incidental. They were based on the belief that diabetes mellitus was purely a disorder of carbohydrate metabolism.

Wherever undernutrition was faithfully carried out, the incidence of coma was reduced and patients lived longer. Only the mild obese diabetic, however, did well. Other adults and children lived very restricted lives and the latter did not grow. Some very striking photographs of pre-insulin

patients are given by Allen (1922).

Present conceptions of pituitary depression associated with starvation states illumine the mechanism of the Undernutrition Principle.

The anterior pituitary gland is thought to produce several hormones including the growth and thyrotrophic hormones, the diabetogenic and corticotrophic hormones and the gonadotrophic and lactogenic hormones. Depression of its activity increases the sensitivity to insulin by reduction of the diabetogenic and corticotrophic hormones, and thus alleviates the diabetic state.

That starvation in normal people produces pituitary depression is clearly recorded by Jacobs (1948). He gives a vivid description of loss of weight, loss of libido, the absence of nocturnal emissions, the thinning of hair on the head, face, axillae and pubis and the atrophy of sebaceous glands, occurring in prisoners-of-war in Japanese camps, subjected to prolonged undernutrition. A decrease in 17-ketosteroid excretion is recorded by Landau et al. (1948) resulting from experimental starvation in human volunteers.

The effect of starvation in lessening the severity of diabetes was noted during the siege of Paris by Bouchardat and in Germany during the first world war. Young (1949) records similar effects in

Germany following the recent war.

Undernutrition lessens the severity of the diabetic state but, because of poor nutrition, the resistance to tuberculosis is lowered. Adults may become thin and tired. Growth and sexual development are often retarded in children.

While undernutrition therapy is of undoubted value in the obese diabetic and cannot be disregarded in diabetes of great severity, the necessity or the desirability of its use in diabetic children or non-obese adults requiring insulin is open to question.

Chapter IV.

THE DEVELOPMENT OF MODERN METHODS.

Following the discovery of insulin by Banting and Best (1922), the old-established principles of undernutrition and normoglycaemia maintained their pride of place in treatment and for many years insulin was used merely as an adjunct to such treatment and in as small doses as possible.

Nevertheless, more liberal methods were foreseen by Allen (1922). Insulin had introduced the possibility of nourishing diabetics and, with the higher diets and doses of insulin required, the margin between hyperglycaemia and insulin reactions became narrow. Allen concluded that to obtain nutrition and at the same time freedom from reactions, a certain degree of hyperglycaemia had to be permitted.

He recognised the calorie demands for growth and development in children and working capacity in adults and the necessity for high nutrition in the treatment of tuberculosis. He was fully conscious of the human element in the adherence to a strict dietary régime. "The diet is not conducted like that of a dog in a cage, where the sole consideration is the accurate proof of a principle."

Three possible forms of treatment emerged.

(i) A strict dietary régime, supplemented by insulin, aiming at a certain degree of undernutrition and the maintenance of normoglycaemia.

(ii) A more liberal treatment permitting full nutrition and allowing mild hyperglycaemia and glycosuria in order to prevent insulin reactions.

(iii) A free method of dieting with complete disregard of glycosuria so long as clinical criteria were satisfied.

Allen favoured the first method and a continuance of undernutrition of sufficient degree to keep the insulin dosage within very moderate bounds. He thought lax régimes might lead to an increase of pyogenic infections and the more frequent and earlier occurrence of degenerative complications. He concluded, "The question may be decided largely by the incidence and mortality of complications under the different methods; and if the different workers will collect and publish strictly objective figures without bias in favour of their own procedures, some trustworthy decision should be possible within the next few years."

Allen's diets for adult diabetics in 1922 consisted of 100-150 grammes carbohydrate, 100 grammes

protein and 111 grammes fat, yielding up to 2000 calories per day. Lawrence quotes his standard diet in 1924 as 50 grammes carbohydrate, 75 grammes protein and 150 grammes fat, yielding 1850 calories. Before 1927, Joslin never used diets containing more than 100 grammes carbohydrate daily.

However, disproportionately high fat diets gradually fell into disfavour as higher carbohydrate intakes were advocated by Adlersberg and Porges (1926), Sansum et al. (1926), Gray and Sansum (1933), Geyelin (1926 and 1935) and Rabinowitch (1930, 1935, 1944).

Among the advantages claimed by these writers was the greater psychological and physical wellbeing of the patients. Diets were less frequently broken and the lower fat content made them more digestible. Hunger was avoided and reactions were few. There was no increase in hyperglycaemia and little if any extra requirement of insulin. The susceptibility to pyogenic infections and tuberculosis was reduced. Lower blood cholesterol levels were thought to have a beneficial effect on the cardiovascular system.

Carbohydrate-fat ratios of 2, 3 or 4 to 1 were advocated and Rabinowitch gave ratios of 6 to 1.

Nevertheless, subcaloric diets were used by all the writers except Geyelin who believed in normal nutrition and whose diets were similar to those of

the general population except that they were regulated. He not only discarded high fat diets but the undernutrition principle as well.

Finally, a free diet school has sprung up which allows normal unregulated diets to diabetic patients.

Controversy rages at the present time as to the best diet to prescribe for the non-obese adult diabetic requiring insulin. Some writers still advocate undernutrition. In certain centres high fat-low carbohydrate diets are used; in others, normally proportioned diets. A minority of physicians allow free diets.

The following table summarises the diets recommended by various writers.

	Author	Daily Carbohydrate Grammes	Daily Fat Grammes	Daily Protein Grammes	Daily Calorie Intake
1.	Dunlop	160	137	98	2265
2.	Geyelin	282-500	60-90	74-120	1964-3290
3.	Joslin	150	80	70	1600
4.	Lawrence	150+	90	75	1710+
5.	Rabinowitch	275	45	80	1800
6.	Wilder	167	178	84	2606
7.	Normal Diet	200-500	80-120	70-100	1800-3480
8.	Present Series	217-551	60-120	67-136	1880-3748

Sources of Information in Table.

1. Textbook of Medical Treatment, edited by D. M. Dunlop, L. S. P. Davidson and J. W. McNee, Edinburgh, 1949, fifth edition, page 348.

2. Geyelin, H. R. (1935), J.A.M.A., 104, 1203.

3. The Treatment of Diabetes Mellitus, edited by E. P. Joslin and others, London, 1947, eighth edition, page 259.

4. The Diabetic Life, by R. D. Lawrence, London, 1947, thirteenth edition, page 39.

5. Rabinowitch: quoted by Joslin, The Treatment of Diabetes Mellitus, page 352.

6. Clinical Diabetes Mellitus and Hyperinsulinism, by R. M. Wilder, Philadelphia and London, 1941, page 118.

7. Normal diet. The Diabetic Life, by R. D. Lawrence, page 39.

8. Present Series. Full details of the diets chosen are given in the appendix.

Comment on Table.

Undernutrition is advocated by Dunlop, Joslin, Lawrence and Rabinowitch. Geyelin and Wilder give normal calorie diets. Dunlop and Wilder use fat as a major source of calories, whereas Rabinowitch believes in very low fat diets. Geyelin, Joslin and Lawrence give normal fat contents. Geyelin's diets differ from free diets only in that they are regulated. The diets chosen by patients in the present investigation are normal in total calories and in the relative proportions of carbohydrate, fat and protein.

Arguments for and against undernutrition have already been discussed.

Fat is slowly absorbed from the intestine and there is delay in the formation of blood sugar from the keto-acids derived from fat. For these reasons, hyperglycaemia and glycosuria are less marked after a fatty meal. The present British rationing system allows a triple fat ration to diabetics which enables relatively high fat diets to be used, and they are widely advocated.

However, there are several points in favour of normally proportioned diets. Their lower fat content allows easier digestion. Recent work has shown the vital rôle played by the liver in carbohydrate

metabolism and the detrimental effects on the liver of high fat diets. It would appear rational, therefore, not to give a diabetic more fat than is usually consumed by a normal person. Joslin avoids high fat diets because of the danger of ketosis.

For example, the diet of 160 grammes carbohydrate, 137 grammes fat and 98 grammes protein with a total of 2265 calories, has a carbohydrate-fat ratio of 1.2 to 1. If this is altered to 3 to 1, keeping the protein and total calories practically the same, the diet becomes 270 grammes carbohydrate, 90 grammes fat and 98 grammes protein, and 2282 calories. This allows a considerable increase in the daily bread ration so that the patient feels less restricted.

Rabinowitch advocates very low fat diets indeed in order to protect the liver and also because he believes such a method lowers the incidence of hypercholesterolaemia and cardiovascular disease.

On the other hand, a certain minimum intake of fat is required to supply fat-soluble vitamins and certain fatty acid radicles which cannot be manufactured in the body de novo. The exact figure for this minimum is not accurately known.

Free diets provide normal calorie intakes and normal proportions of the various foodstuffs. They differ from all of the foregoing in that they are

unweighed and no estimation of food quantities is made whatsoever.

Chapter V.

FREE DIETS.

Advocates of free diets pay little attention to glycosuria so long as clinical standards of control are satisfied. Successful treatment of diabetes mellitus should ensure the following fourteen criteria:

1. Freedom from thirst.
2. Freedom from symptoms of polyuria.
3. Freedom from pruritus.
4. Freedom from acetonuria, ketosis and coma.
5. Full physical and mental energy.
6. The attainment and maintenance of an adequate weight.
7. The avoidance, on the other hand, of obesity.
8. Adequate growth and
9. Normal sexual development in children.
10. Good nutrition and resistance to tuberculosis and
11. Freedom from repeated pyogenic infections.
12. Freedom from hunger.
13. Freedom from insulin reactions, and, finally,
14. A happy psychological outlook.

Whereas practically normal fertility may be attained by the treatment of diabetes in women, the foetal loss rate remains high.

One should like to add freedom from degenerative complications to the above list, but no means of achieving this ideal has been discovered.

Freedom from thirst, polyuria and pruritus depend upon good diabetic control in the sense of a low, or at least asymptomatic, level of glycosuria.

Freedom from ketosis, full physical energy, the maintenance of an adequate weight, normal growth and development and good resistance to tuberculosis and probably to pyogenic infections, are attained by adequate carbohydrate utilisation.

Full diets prevent hunger and, when not in excess of the individual requirement, avoid obesity.

Reactions may be minimised by suitable distribution and dosage of insulin.

A happy psychological outlook is promoted when good clinical control is achieved with the minimum restriction on the patient's activities.

Undernutrition is the simplest way of reducing glycosuria but it may lead to hunger and an inadequate carbohydrate utilisation for optimal health and strength. Regulated normal calorie diets should ensure excellent clinical control in non-obese adults.

Free diets have an additional psychological advantage.

Following the discovery of insulin, the possibility of doing away with severe dietetic restriction was recognised. However, Allen and Joslin did much to maintain rigid dietetic control in America.

In Europe originated the free diet method, which was begun independently by Stolte of Breslau (1931) and Lichtenstein of Stockholm (1938, 1945).

Many other European writers advocated liberal régimes, including Söderling of Sweden, Raïhä of Finland, Sundahl of Norway, Bojlén of Denmark and Ercklentz, Frick, Musterle, Müller and Kestermann of Germany.

Tolstoi (1939, 1940, 1942, 1943) of America and Micks (1943, 1944) of Ireland are more recent writers on the subject.

The work of Stolte, Lichtenstein, Tolstoi and Micks will be considered in detail.

Stolte's article on the treatment of children was the first to appear in praise of free diets. He described the gloomy prognosis of juvenile diabetes before insulin and the tremendous benefit its discovery brought about. However, even with diets higher than had ever previously been possible, acidosis was common and clinical results did not please him.

He gradually introduced more carbohydrate into his diets and found the children correspondingly

improved. Then, with a view to reducing the psychological reaction to dietary restrictions, he tried free diets with astonishingly good results. The method suited younger and older children with mild or severe diabetes. Insulin requirements were greater than on controlled diets but not to the extent expected from the rise in carbohydrate consumption. His diets contained approximately 200-400 grammes of carbohydrate and up to 100 grammes of protein and fat.

Three injections of insulin were given daily, one before each main meal. No attempt was made to avoid glycosuria.

On sensible diets, avoiding over-indulgence in sweet things, his results were good. The children were happy, healthy and full of energy.

Stolte felt, however, that a long time must elapse before definite conclusions could be drawn as to the final success of the treatment.

Lichtenstein wrote with a background of thirty-five years' experience of diabetic treatment. He had been working for fifteen years before the discovery of insulin and for the ensuing ten years had used strict diets and insulin. Thereafter, ten years' experience of a free diet method left him in no doubt as to its superiority. He was particularly

impressed by the greater happiness and well-being of the children and claimed the attainment of excellent clinical control.

169 children were followed up for ten years with only 8 deaths, and a low incidence of complications. Children were given a free choice of foods but the mothers were instructed to avoid over-feeding them, and to discourage greediness. Sweets were allowed, in moderation. Diets averaged from 100-250 grammes carbohydrate daily, with normal proportions of protein and fat. A single daily dose of Hagedorn's protamine insulin, without zinc, was employed, varying from 10 to 120 units. Close cooperation with the mothers ensured regular meals and injections. Sugar and acetone tests were done frequently. Acetone was always absent unless in emergencies and a twenty-four hour sugar loss of less than 10% of the carbohydrate intake was the rule. This low level of glycosuria is remarkable on a free diet régime in the absence of reactions. Fasting blood sugar levels varied between 100-200 mg. %. Growth and sexual development were very satisfactory and it is notable that no difficulty with the method was encountered during the years of puberty.

The major American writer to challenge the citadel of purism held by Joslin was Tolstoi. He

employed unweighed diets of approximately 200-300 grammes carbohydrate and symptoms were controlled by a single daily dose of zinc protamine insulin. A maximum total of 60 units was used. It is remarkable that reactions were few when such a method was used. A five year follow up proved that good clinical results were maintained despite glycosuria of "much more than 100 grammes in the twenty-four hours".

Tolstoi's view was that emphasis should be placed not on sugar-free urine but on adequate carbohydrate utilisation. This conception is so important that a digression will be made to consider a few very simple biochemical points.

Himsworth (1949) defines the one essential for the syndrome of diabetes mellitus as an elevation of the blood sugar level above what is commonly accepted as normal, with resultant glycosuria, polyuria and thirst. On the other hand, Peters and Van Slyke (1931) consider diabetes mellitus to be a condition in which the maximum attainable rate of carbohydrate combustion is reduced below normal and the ability to store glycogen is lost. No mention is made of hyperglycaemia.

Liver glycogen is formed directly from carbohydrate in the diet and may be synthesised also from a

pool of keto-acids derived from protein and fat (Soskin and Levine, 1946). The liver pours glucose into the systemic circulation from which muscle glycogen reserves are built up. The combustion of these reserves produces energy for work. Good liver glycogen stores imply adequate carbohydrate for oxidation, prevent ketosis and protect the vital parenchymal cells. Physical energy is dependent upon good muscle glycogen stores.

A normal blood sugar concentration may exist in the presence of low levels of liver and muscle glycogen when sub-caloric diets are used. Hyperglycaemia, on the other hand, by no means necessarily implies inadequate stores, unless there is associated ketosis.

In the production of energy, the body draws first upon its carbohydrate stores, then on fat and, finally, upon protein. Starvation, or the inability to utilise carbohydrate, as in untreated diabetes, leads to excessive demands on fat metabolism and consequent ketosis. The use of protein for energy purposes finally produces a negative nitrogen balance and loss of flesh.

Treatment of diabetes should ensure that adequate carbohydrate can be utilised so that ketosis and loss of weight may be avoided.

Micks was so satisfied with the free diet treatment of diabetes that all his diabetics, children and adults, received this form of treatment. The only exceptions were patients with diabetes of recent onset who were carefully controlled in an attempt to produce remission, but were given free diets once it was clear that the diabetes had become established.

Diets were free except for regulation of meal times and advice was given regarding snacks to be taken between each meal. He noted that the dietary carbohydrate contents were high and the protein often low. One would prefer, however, an optimal protein intake for diabetics.

Symptoms were controlled by two doses of soluble insulin in the day for adults and three doses for children. The largest dose was in the region of 60 units daily.

The patients were taught no urine tests and hyperglycaemia and glycosuria were disregarded. He quotes twenty-four hour urine outputs as 50 oz. - a remarkably low figure for glycosuric patients.

Clinical results were excellent; over an eight year period the patients exhibited the maximum degree of health and vigour.

Free diets in the treatment of diabetes mellitus are open to two major criticisms.

Firstly, the quality of the diets chosen may not

be ideal for optimal nutrition. Joslin (1947) teaches that the diet of a diabetic should be equal if not superior to the normal diet, properly balanced, and adequate in vitamins and minerals of which vitamin B and salt are particularly important. Wilder (1941) thinks free diets may be inadequate in protective foods. There is no necessity to advise weighing of foods to ensure diets of good quality. A few simple instructions are all that is required.

Secondly, the quantity of food may be in excess of the patient's needs and lead to obesity. The experience gained by this survey suggests that the danger of obesity exists only in those who have been over-weight in the past.

That unregulated diets lead to hyperglycaemia is undoubted. The important question of the possible harmfulness of hyperglycaemia is considered in a later chapter.

The writers on free diets show minor individual variations in clinical method, the most notable being in the use of different insulin preparations. The present survey limits concentrated carbohydrates and employs a more complicated combination of insulins to give optimal control while at the same time reducing the danger of reactions.

The fundamental conception is the same, however,

in all methods. Patients are allowed maximal freedom in their daily lives, and insulin is given to promote clinical well-being, little regard being paid to glycosuria provided it is symptomless.

Chapter VI.

PSYCHOLOGICAL ASPECTS OF TREATMENT.

Diabetes Mellitus is a chronic disease, and, although with insulin therapy the patient may be offered a very reasonable life, no cure is possible. Insulin injections, dietary regulations, and the danger of reactions or ketosis are constantly reminding the diabetic of his disability. It is not surprising, therefore, that psychological repercussions occur from time to time, even in the most robust personality. From this point of view, the fewer restrictions and regulations imposed upon the patient the better.

Quite a few doctors, themselves diabetic, have written about diabetes. The irregular hours of medical practice are not conducive to a régime of normoglycaemia and many doctors follow a more or less liberal régime, welcoming a degree of glycosuria as a safeguard against unpleasant reactions.

A Doctor, writing in The Practitioner (1946), described his horror when he realised he had diabetes. "In future no meal would be an elegant satisfaction of appetite, but a problem in arithmetic and a trial of self-negation. No longer would I live my life

at the behest of work and leisure, but to a clockwork routine of injections and measured meals. Holidays, I read, were times of special risk. Wherever I went I should be encumbered by the paraphernalia of urine testing, balances, weights and measuring glasses."

Another Doctor described his twenty-three years' experience of diabetes in The Lancet (1949). In 1925, according to the custom of the times, he was given a rigid, high fat-low carbohydrate diet and two doses of soluble insulin daily. Gradually he became more liberal in his dietary habits and, at present, takes normal meals, avoiding only concentrated carbohydrates, and he shares his extra fat ration with his family. The most disquieting feature of his diabetic life has been the incidence of reactions which may be largely due to irregular hours of work in general practice. Nevertheless, on looking back, he does not consider his diabetes to have been a great disability and, since taking a good full diet, his strength and energy have been remarkably good.

Adult diabetics may develop at least a superficial adaptation to dietary restrictions but the ill effects of these on children are more obvious.

Fisher and Dolger (1946) gave an excellent survey of the behaviour and psychologic problems of young diabetic patients. The normal temporary

alterations in behaviour in the transition from childhood to adult life they considered to be more pronounced in diabetic children because of the abnormal regimentation to which they are subjected and because of certain psychic and physical factors associated with their condition.

Whereas a child living at home may be easily looked after, if tactfully handled by good parents, school and play groups demand a personal adaptation from the young diabetic. To permit full physical activity, the writers prescribed higher diets. The children themselves volunteered that they felt fitter with glycosuria than without.

When schooldays are over, the young person must face contact with the outside world. Young men do not wish to feel handicapped in a competitive environment, and young women are anxious lest their diabetes interferes with marriage and the possibility of having children.

The writers stressed the importance of avoiding reactions, which, apart from causing fear, produce prolonged electro-encephalographic changes and lead over many years to mental deterioration. They believed, however, that behaviour disorders were largely due to diabetic regimentation.

Loughlin and Mosenthal (1944) described their

experience in a summer camp for diabetic children. Of these, 40% had abnormal personalities. With relaxation of the strict methods, improvement occurred and the best method was found to be the use of unweighed diets, avoiding concentrated carbohydrates, with urine testing reduced to a minimum.

They concluded: "The child is indeed fortunate who is treated at a clinic by a doctor who can control the diabetic condition without instilling ever present consciousness of calories and carbohydrates, of urine analysis and of injections of insulin. The child who is different from his playfellows is a good candidate for neurosis."

A quotation from the Editorial of The American Journal of Digestive Diseases (1949) reads:

"There is every reason to think that Tolstoi should be considered right until he is proved wrong. The effort at perfection in maintaining constant (?) blood sugar levels of a normal range involves the frequent sampling of blood and such strict adherence to a meticulous food intake that life is rendered a great burden to the patient who often rebels and goes berserk. Actually the treatment of any diabetic, viewed in retrospect, is seen to be a record of hope and discouragement, of discipline and license, of resignation and rebellion, of success and failure

and usually of final engulfment in that mysterious sea of degenerative processes which causes death. Tolstoi at least offers the individual a grateful degree of freedom as he pursues his course."

A similar point of view is expressed in the Running Commentaries by peripatetic correspondents in The Lancet of March 5th and 12th, 1949. The danger, inherent in complicated régimes, of inducing a pre-occupation with diabetes is emphasised. "The most important thing about any ailment is the patient's attitude towards it, and the extent to which the ailment is allowed to interfere with the patient's normal life and activities. A diabetic who lives for his diabetes, however correctly and successfully, leads a worthless life compared with the patient who ignores it and goes about his business unconcernedly."

These extracts provide an amplification of the psychological factors involved in the treatment of diabetes. Writers advocating liberal dietetic régimes claim a minimum of psychological repercussion. Such free methods offer patients a life much closer to normality and it is this undoubted advantage which prompted the following investigation.

Chapter VII.

THE PRESENT INVESTIGATION.

1. SELECTION OF PATIENTS.

Fifty-five patients were given liberal diets and insulin over a period, covered in this survey, from October 1945 to March 1949. For purposes of comparison, forty patients were studied from the Dietetic Clinic. The work was done in Ward 21 of the Royal Infirmary, Edinburgh, under the supervision of Professor D. M. Dunlop.

In the selection of patients for a liberal régime, the obese were purposely omitted. Obese diabetics are usually fat women who often do well on dietary restriction alone without insulin. Because of the exclusion of the latter more men than women were chosen. Twenty-eight new diabetics were included at random, as they appeared for stabilisation. Twenty-seven previously treated diabetics were also taken over, who came after changing their home town and were not following a dietetic régime, or who had been attending the Dietetic Clinic but were making no effort to adhere to dietary instructions.

The free diet group finally included thirty-five men and twenty women of ages ranging from nine to seventy-one years, at the commencement of treatment. They are listed in the appendix where full details of the individual patients are given.

For comparison, forty patients were selected from the Dietetic Clinic. All were taking insulin and adhering to their diets. Otherwise, the selection was random, and, finally, comprised seventeen men and twenty-three women of ages ranging between thirteen and sixty-seven years. This group represented a cross section of the careful, regularly attending, diabetics of the Clinic and did not take into account the defaulters, who were by no means few. Their control was as good as could be obtained in co-operative patients attending a busy diabetic centre. Their data is also given in the appendix.

Except for five patients of the free diet group, all were uncomplicated cases of diabetes so far as could be assessed from careful clinical examinations, chest X-rays, blood counts, W.R. and B.S.R. investigations.

In the collection of data for presentation, owing to the practical difficulties attendant upon all clinical research, certain patients had to be omitted for a variety of reasons, including other complicating illnesses, temporary changes of address, hours of work making the collection of blood and urine samples impossible and poor co-operation on the part of a few patients. The numbers of patients included in the various surveys therefore differ, but it

is emphasised that omissions were in every case due to the above obstacles and that no selection was employed.

The blood sugar estimations and the urine sugar estimations were done by myself by the methods of Hagedorn and Jensen, and Cole respectively, given on pages 343, and 297 of Clinical Chemistry in Practical Medicine by C. P. Stewart and D. M. Dunlop, second edition, Edinburgh, 1937.

2. METHOD OF TREATMENT.

The fifty-five patients of the free diet group were allowed to eat as they pleased except for the omission of concentrated carbohydrates in the form of sugar, jam and sweets. There was no weighing or estimation of food quantities whatsoever. By choice, the majority of patients took three main meals with a snack in the middle of the morning and before going to bed at night.

During the period studied, the British rationing system allowed diabetics extra meat, bacon, milk, cheese and fats. As the patients availed themselves of these extras, the standard of nutrition was higher than that of the general population.

Simple dietary instructions to ensure an adequate intake of protein and vitamins might be required in a non-rationed community. One helping of meat, one helping of fish or an egg, one ounce of cheese, one pint of milk and one helping of fresh green vegetables or fruit, taken daily, ensure a good diet.

Every patient received insulin which was employed twice daily in most instances, half-an-hour before breakfast and half-an-hour before the main evening meal. Stabilisation was effected in hospital. The usual method was to give an injection of zinc

protamine insulin and soluble insulin in the morning and a dose of soluble insulin in the evening.

The patient was made thoroughly conversant with insulin reactions before leaving hospital and with the value of sugar in alleviating them. On discharge, lump sugar was always carried to ward off reactions. Slight modification of insulin dosage and the prescription of an extra snack in the morning were sometimes required to suit working conditions.

Patients were taught to test a morning specimen of urine for acetone twice weekly as a routine or every day during infections such as colds, and to report immediately if acetone were present. A simple powder test devised by Mr John Ingram of the Welsh National School of Medicine, Cardiff, is used as follows:-

℞ Sodium Nitroprusside 3 G.
Ammonium Sulphate 100 G.
Sodium Carbonate Anhydrous 50 G.

Make a powder.

Sig. Acetone Testing Powder for use as directed.

Patients place about half an inch of powder in a small test-tube and wet it with a few drops of urine. A purple colour indicates that acetone is present. There is no colour change in the absence of acetone. The acetone test should always be negative and this gives the patient confidence.

A monthly visit to Ward 21 enabled constant supervision of the progress of each patient. Each time the weight was checked, and any insulin reactions, nocturia, positive acetone tests, symptoms or complaints were noted down.

No instructions were given regarding tests for urine sugar by the patients themselves but at each visit two urine samples were brought for quantitative analysis of the sugar content:

(i) A morning sample.

This was obtained by emptying the bladder on rising and then collecting the sample passed twenty minutes later before food or insulin were taken.

(ii) A twenty-four hour sample.

The collection was made the previous day and its measurement written down in pints. A small quantity was brought for estimation.

The dose of insulin was adjusted to maintain freedom from acetone, an adequate weight and complete clinical control.

3. FAMILY HISTORY.

Of the ninety-five patients studied, twenty-four had a positive family history of diabetes mellitus, giving an incidence of 25%.

4. OCCUPATION.

Occupations varied greatly in both groups and involved a wide range of physical exercise, from that of the sedentary clerk to that of a dock labourer.

The occupations of the free diet group of fifty uncomplicated diabetics are given below.

M E N	
Occupation	Number
At school	2
At university	1
Unemployed	2
Labourers	7
Miners	6
Tradesmen	8
Business Men	8
Lorry Drivers	3

W O M E N	
Occupation	Number
Housewives	11
Typists	2
Shop Assistants	2
Business Women	1

The lorry drivers were advised to change their job, but were unwilling to do so. Otherwise only one patient gave up his work for something else. He was employed on a deep sea trawler and decided to change to labouring so that he could be at home for his insulin injections and meals. The dietary restrictions in a rigid régime make a change of occupation necessary more frequently than the liberal method.

5. INTELLIGENCE AND CO-OPERATION.

On the whole, better results were obtained by intelligent, co-operative diabetics in that they had the upper hand of their disability and were quick to spot incipient reactions or the early symptoms of poor control.

The majority of the free diet group were at least of average intelligence and quite co-operative. However, the successful results in the other rather scatter-brained minority were remarkable.

6. INSULIN.

Insulin was discovered by Banting and Best (1922) in a form which could be used clinically without producing violent side effects. The earliest preparation was soluble insulin.

In 1936, Hagedorn produced a new preparation called protamine insulin whose effect had a slower onset and was more prolonged. Zinc protamine insulin has an even longer action.

Reiner (1939) developed globin insulin with a duration between that of soluble and zinc protamine insulin.

The time-activities of single large doses of various types of insulin in diabetes of moderate severity are given below in a table from Colwell (1947).

	Type of Insulin	Action Demonstrable	Peak Action	Intensity at Peak	Duration of Effect
		Hours	Hours		Hours
1	Soluble Insulin	1	3-6	Strong	8-12
2	Globin (with Zinc)	2	8-12	Fairly strong	24 maximum
3	Zinc Protamine Insulin	4-8	24-32	Weak	3 days or more

With free diet treatment, the type of insulin used and its distribution to achieve the best results varied from patient to patient, the individual details being given in the appendix.

As a general rule, however, the method adopted was that of a dose of soluble insulin and zinc protamine insulin in the morning - not mixed in the syringe - and a dose of soluble in the evening. Approximately equal doses of each were given, e.g. 16 units of soluble + 16 units of zinc protamine insulin in the morning and 16 units of soluble insulin in the evening. Injections were taken half-an-hour before breakfast and half-an-hour before the evening meal.

Forty out of the fifty free diet patients received two daily injections.

This method of using insulin combines the prolonged effect of zinc protamine insulin, without its dangers of insidious reactions, with the flexibility obtained by two doses of soluble insulin.

7. DIETS.

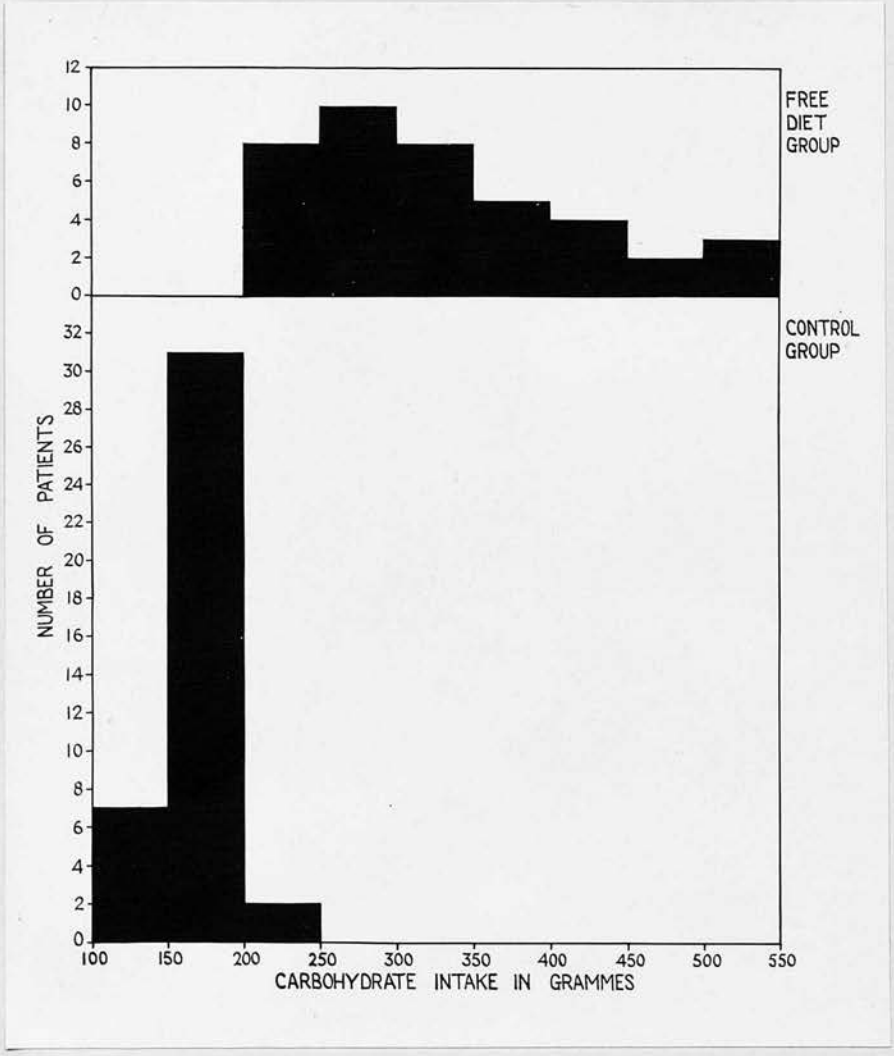
Patients on a free diet were allowed to eat what they liked except for jam, sweets and raw sugar. They were instructed to take a good meal following their insulin injections and many were advised to have a mid-morning snack.

This restriction prevents overloading of the carbohydrate regulatory mechanism and, particularly, avoids wild and unnecessary fluctuations of intake. It amounts to a reduction of about 100 grammes of concentrated carbohydrate daily.

The majority adopted a régime of three main meals, namely, breakfast, lunch and tea, with snacks in the morning and before going to bed. Timing varied with hours of work but remained fairly constant from day to day.

Those patients on free diets whose co-operation and accuracy could be trusted were asked to write out everything they ate or drank for a period of one week. None of the patients possessed scales, but great care was taken to make the estimation of the diets as accurate as possible. The details of the method of assessment are given in the appendix.

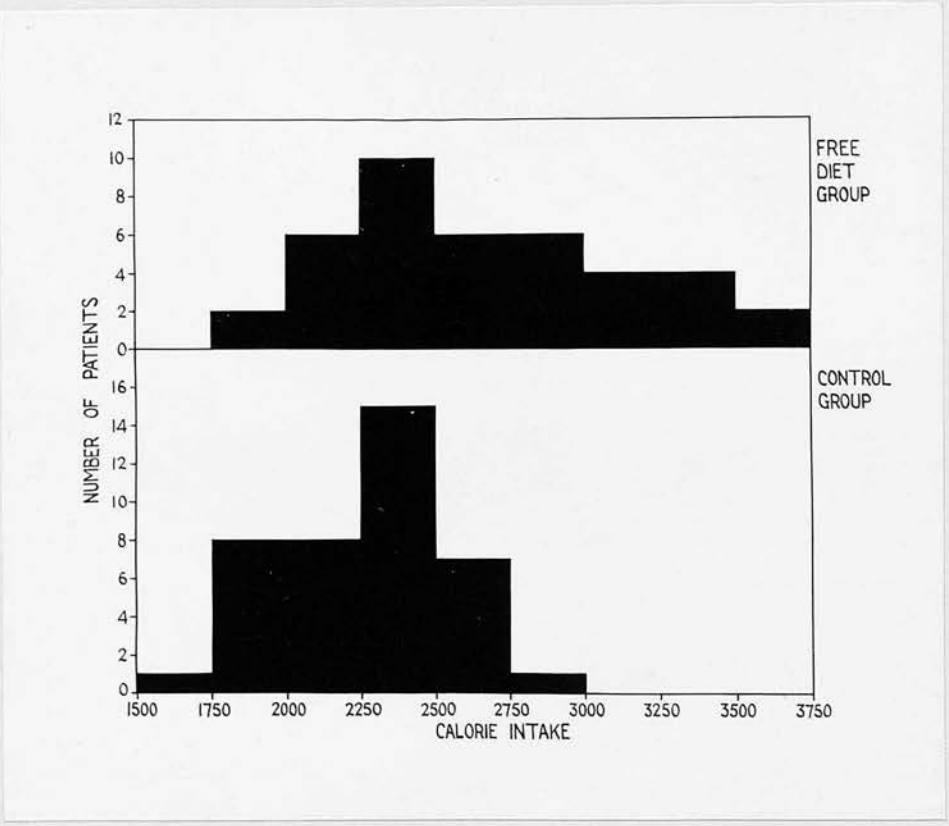
Graph 1.



Average Daily Carbohydrate Intakes of the Free Diet Group compared with those prescribed for the Control Group.

Free Diet Group - 40 patients
Control Group - 40 patients

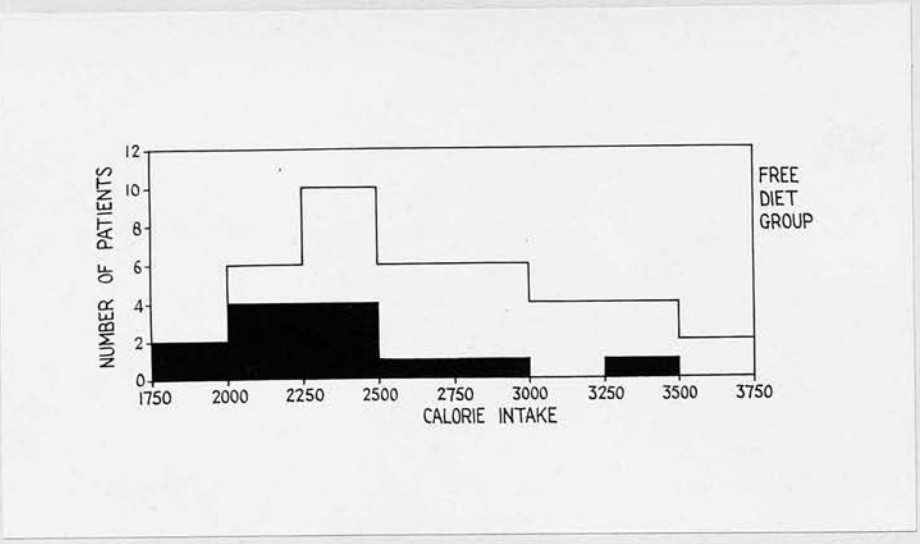
Graph 2.



Average Daily Calorie Intakes of the Free Diet Group compared with those prescribed for the Control Group.

Free Diet Group - 40 patients
Control Group - 40 patients

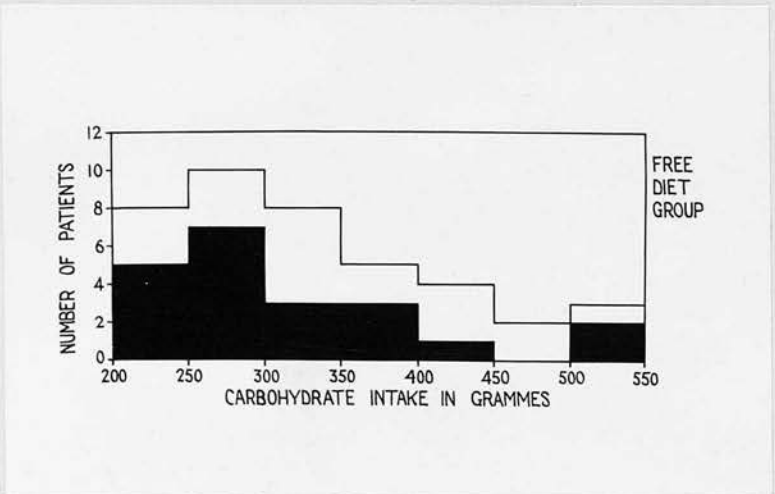
Graph 3.



Average Daily Calorie Intakes of women on Free Diets compared with those of the Free Diet Group as a whole.

Free Diet Group - 40 patients - outlined
Women - 13 patients - in black

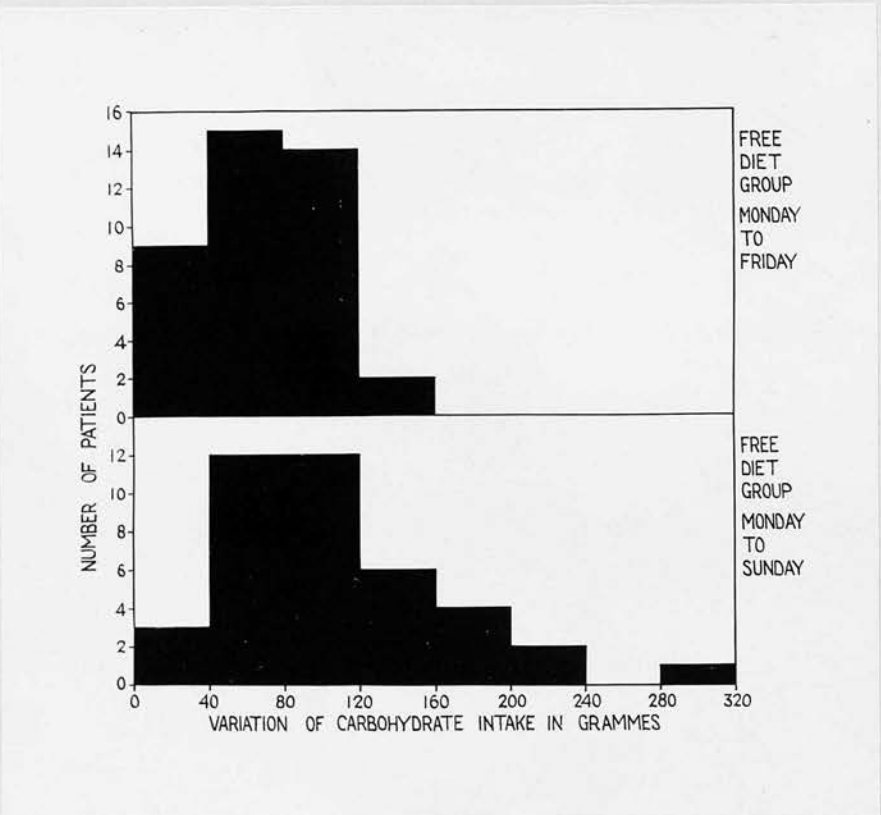
Graph 4.



Average Daily Carbohydrate Intakes of previously treated diabetics compared with those of the Free Diet Group as a whole.

Free Diet Group - 40 patients - outlined
Previously treated diabetics - 21 patients - in black

Graph 5.



Variation of the Daily Carbohydrate Intake from Monday to Friday compared with that from Monday to Sunday.

Free Diet Group - 40 patients.

Graph 1.

The carbohydrate intakes of patients on free diets are considerably higher than those prescribed in the Dietetic Clinic.

The variation of intake from patient to patient is also much greater when free choice is permitted.

Graph 2.

The calorie intakes of the free diet group are also higher and the individual variation greater than for the control group. However, the difference is not quite so marked because of the larger proportion of fat in the controlled diets.

Graph 3.

The diets chosen by women on a free régime are on the whole less than those of the men.

Graph 4.

The carbohydrate intakes of diabetics who had been previously treated before being given free diets do not differ significantly from those taken by new diabetics. In the selection of previously treated patients only those who were by no means following their dietary instructions were included and many were taking a free diet faute de mieux, as co-operation had not been achieved.

Graph 5.

The variation of carbohydrate intake from day to day on a free diet is not nearly so great during weekdays under working conditions as at weekends.

It was found that heavy labourers who enjoyed a lazy weekend were not so hungry on Saturday and Sunday, whereas sedentary workers who played golf or indulged in other forms of exercise at weekends ate correspondingly more. A few patients required a change of insulin at weekends to suit the altered conditions.

Summary.

Patients allowed to choose their diets eat more than is prescribed on a weighed diet system and the variation of intake from patient to patient is greater.

As would be expected, women choose smaller diets than men on the whole. Patients who are much more active at weekends tend to eat correspondingly more at that time and vice versa.

---

8. THIRST, POLYURIA, PRURITUS.

Patients receiving free diet treatment should be given sufficient insulin to prevent thirst. If freedom from thirst cannot be achieved, controlled diets must be used.

Polyuria of some degree is a concomitant of glycosuria. However, polyuria, insufficient to obtrude itself on the patient's consciousness was permitted. Polyuria is most noticeable when it necessitates rising at nights. Normal people may rise once on one night in the week and this degree of nocturia was disregarded. Only two patients rose on at least two nights of the week. The first patient (J.J.) had an average twenty-four hour urine output of 2925 c.c. and his control was admittedly borderline; the other (V.S.) passed only 1950 c.c. in the twenty-four hours and her explanation of nocturia as due to habit was probably correct. The vast majority of patients, despite urinary outputs above what one usually considers as normal, did not experience nocturia or frequency of micturition.

Pruritus Vulvae, like thirst, must be entirely avoided in the treatment of diabetes. Three women (A.A., J.Pa. and H.G.) complained of pruritus vulvae on free diets and were changed to a restricted intake. The rest of the group did not experience pruritus, despite considerable glycosuria.

9. INSULIN REACTIONS.

Almost every diabetic patient, at some time or other, experiences mild insulin reactions. These are quickly forgotten and of very little consequence. However, where reactions are frequent or result in coma, clinical control cannot be considered satisfactory.

Two patients (J.L. and A.N.) had to be given regulated diets because of frequent reactions. Two others (R.Gr. and A.D.) would be better on regulated diets for that reason, but there is no possibility of their adhering to dietary instructions.

Four patients (G.S., A.W., F.Mc. and J.McWh.) have experienced a hypoglycaemic coma. Both G.S. and A.W. were discharged from another ward on a dose of insulin which proved excessive under home conditions. F.Mc. changed to a much heavier labouring job without reporting the fact to the Clinic and the extra exercise resulted in a severe reaction. No cause could be found for that of J.McWh. who continued thereafter on the same dose of insulin without further incident.

The remaining thirty-nine of the forty-seven uncomplicated diabetics whose follow ups were complete, experienced no inconvenience from reactions

despite uncontrolled diets. The method of using insulin previously described may have contributed largely to this satisfactory result.

10. KETOSIS and COMA.

Intercurrent infections induced mild ketosis in several patients who recovered quickly following a temporary increase of insulin. Two patients developed diabetic coma during treatment on a free diet.

A.Mc. became comatose while being treated in bed at home for a severe streptococcal lymphangitis following a septic toe. This patient, a severe diabetic, was subsequently given a controlled diet.

The second patient, G.S., developed diabetic coma following an alimentary upset. Shortly after his discharge from another ward he was readmitted in hypoglycaemic coma. For a few months he remained very well, when he was again admitted in coma and died following massive doses of insulin in another ward.

11. PYOGENIC COMPLICATIONS.

Boils, styes and septic cuts occur from time to time in diabetics. Injection abscesses, however, are usually very rare.

Seven patients of the free diet group developed more than one pyogenic complication in a year. One of these patients and three others developed an injection abscess. Details are given in the following table.

	Initials	Type of Infection	Year
1	B.R.	Injection abscess	1947
2	L.H.	Injection abscess	1947
3	R.McWh.	4 boils	1947
4	J.McWh.	2 boils 1 septic cut + 1 boil	1947 1948
5	H.S.	1 boil + 1 septic cut	1946
6	A.C.	3 boils	1947
7	A.Mc.	2 injection abscesses 1 septic toe + several styes	1947 1948
8	E.Ber.	Injection abscess	1947
9	J.Pa.	1 boil + 1 stye 1 boil + 1 stye	1947 1948
10	C.Wa.	2 styes	1948

The overall incidence of pyogenic complications was not strikingly high. In only one patient out of the forty-seven carefully followed up did sepsis occur frequently. She was A.Mc., a severe young diabetic who had always been subject to pyogenic infections even before developing diabetes. Although changed to a strict diet with a view to reducing glycosuria to a minimum, three series of styes have occurred since the change.

Three patients as well as A.Mc. developed an injection abscess, but in them no other sepsis occurred. That free diets usually necessitate two doses of insulin daily may be a factor in producing the apparently increased incidence of injection abscess.

12. MISCELLANEOUS COMPLICATIONS.

Five patients, receiving a free diet, are listed at the end of the uncomplicated group of fifty in the appendix because of other illnesses associated with their diabetes, namely thyrotoxicosis in two patients and gastric carcinoma, pulmonary tuberculosis and psychoneurosis in the other three respectively. These five patients are excluded from the general survey.

Of the fifty free diet patients included in the data, several had transient complicating illnesses. J.D. and D.W. developed lobar pneumonia, E.H. took cystitis complicating a cystocele, W.T. developed peripheral neuritis following an attack of diarrhoea, C.W. contracted infective hepatitis on a visit to Italy and A.D. was admitted to a mental hospital for a few weeks on account of depression.

13. PREGNANCY.

There was a remarkably high incidence of pregnancy - namely seven out of eight - among women of the free diet group who wished to have children. Four women bore live babies, two had still-births and one is at present pregnant.

The other woman and her husband were anxious to be investigated for the cause of their sterility. The woman has a twenty-two year history of diabetes and her husband on examination was found to be subpotent.

14. DEGENERATIVE COMPLICATIONS.

Peripheral vascular disease, hypertension and albuminuria were not present in any of the free diet patients.

Forty-five were able to report for ophthalmoscopic examination in 1949 and the findings were confirmed by Dr G. H. Scott of the Eye Department. The pupils were dilated with 2% homatropine hydrobromide before examination of the fundus and later the drops were counteracted by 2% pilocarpine nitrate.

Four patients showed diabetic retinitis and one had early senile cataract. Details are given below.

	Initials	Age in Years	Duration of Diabetes in Years	Abnormality
1	R.McWh.	24	12	Early retinitis of Right Eye
2	A.D.	34	8	Early retinitis of Right Eye
3	E.L.	61	2	Early retinitis of both eyes.
4	J.M.	72	1	Early senile cataract
5	W.T.	28	1	Early retinitis of both eyes

The notable patient was W.T. who had retinitis at twenty-eight years after only a year's duration

of diabetes. This patient also exhibited neuritis following an attack of diarrhoea which cleared up.

Mention should be made of the patient, V.S., who has been following a free diet for twenty-two years and has normal fundi. She is now twenty-five.

The duration of treatment is, in most patients, too short for a true assessment of whether hyperglycaemia increases the incidence of retinitis. One may say, however, that so far there is nothing to suggest that the free diet method is altering the prognosis.

15. WEIGHTS.

It is always desirable, in the treatment of diabetes, to avoid obesity, which has been cited as a contra-indication to free diets.

The relationships between the present weight and the correct weight, average weight and highest weight are considered.

Correct weights are obtained from the tables on pages 385 and 367 of the Textbook of Medical Treatment edited by D. M. Dunlop, L. S. P. Davidson and J. W. McNee (fifth edition), Edinburgh, 1949.

The average weight refers to the weight of the patient before he became obese prior to the onset of diabetes, or before he lost weight due to diabetes if there was no history of precursory obesity.

The highest weight recorded in the past clearly allows separation of obese, normal and thin groups prior to the onset of diabetes. This separation is based upon an arbitrary comparison of the highest weight prior to the onset of diabetes and the correct weight. Those who had been more than half-a-stone overweight are classified as precursorily obese, those who had been more than half-a-stone underweight as precursorily thin, and the remainder as normal.

The figures on which the ensuing graphs are based are given in the appendix.

Forty-seven patients in the free group and forty control cases are considered in the charts showing the relation of weight to correct weight.

Thirty-six patients in the free group and thirty-four control cases are considered in the charts showing the relation of weight to average weight, and weight to highest weight. Those who developed diabetes in youth had of necessity to be omitted.

Graphs deal with either averaged weights or absolute weights.

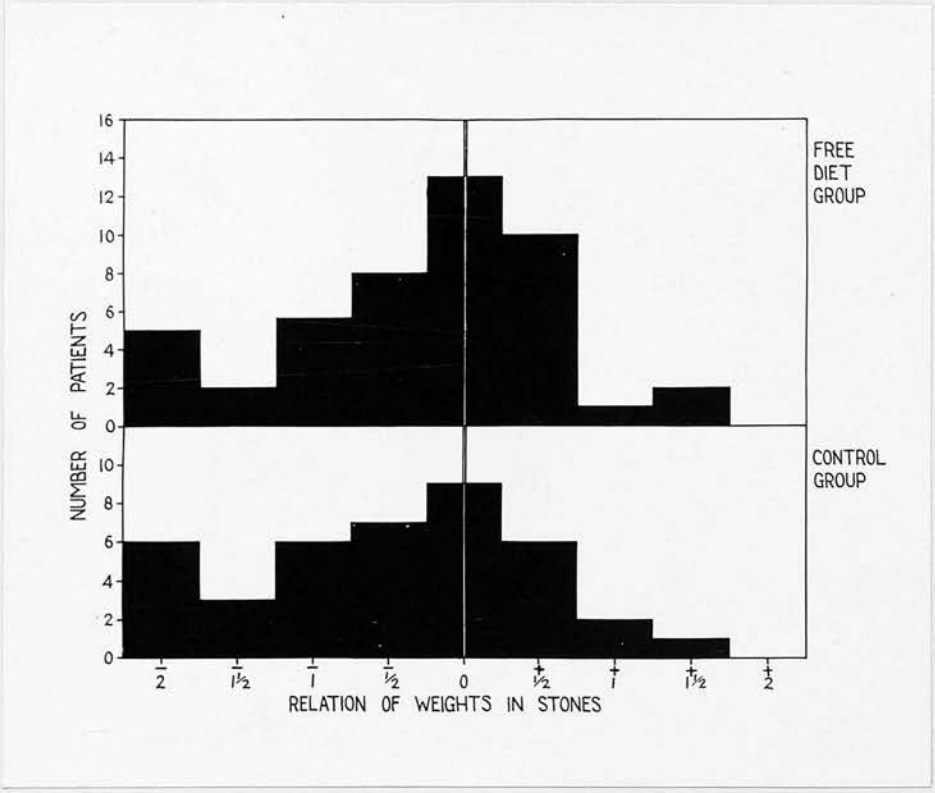
Averaged weights were assessed to the nearest half stone, thus -

0-3 lbs. ≡ 0
4-10 lbs. ≡ ½ stone
11-14 lbs. ≡ 1 stone, etc.

Absolute weights were placed in ranges of half a stone, thus -

0-7 lbs. ≡ 0-½ stone
8-14 lbs. ≡ ½-1 stone, etc.

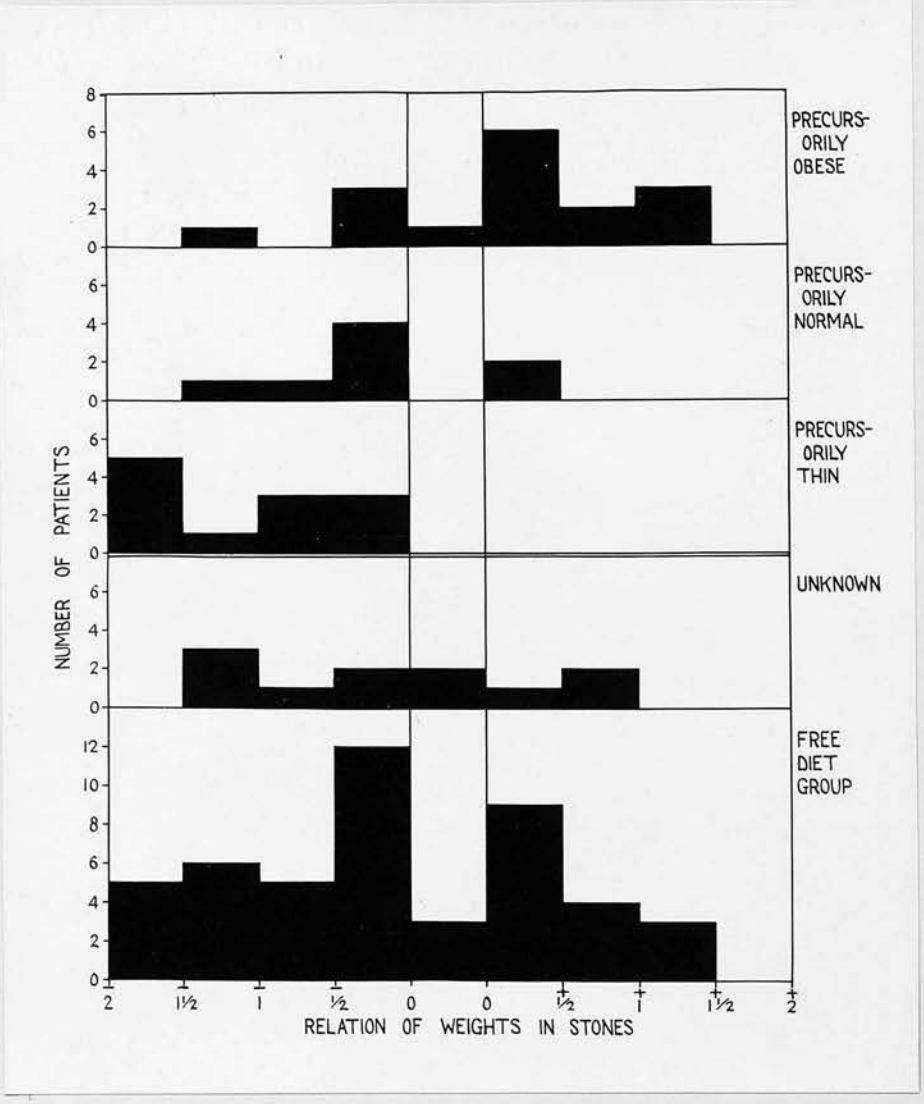
Graph 6.



Relation of weight to correct weight averaged to the nearest half stone.

Free Diet Group - 47 patients
Control Group - 40 patients

Graph 7.

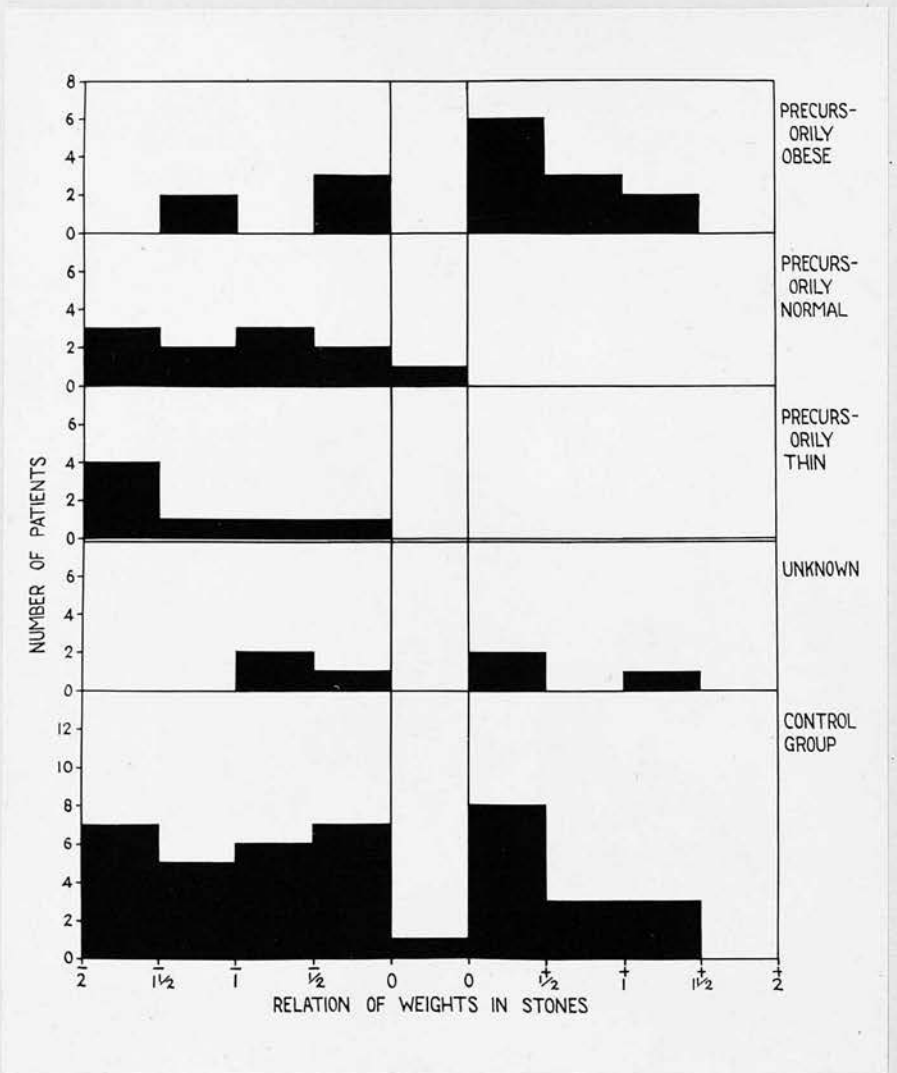


Relation of weight to correct weight - absolute.

Free Diet Group - 47 patients

Precursorily obese	-	16 patients
Precursorily normal	-	8 patients
Precursorily thin	-	12 patients
Unknown	-	11 patients

Graph 8.

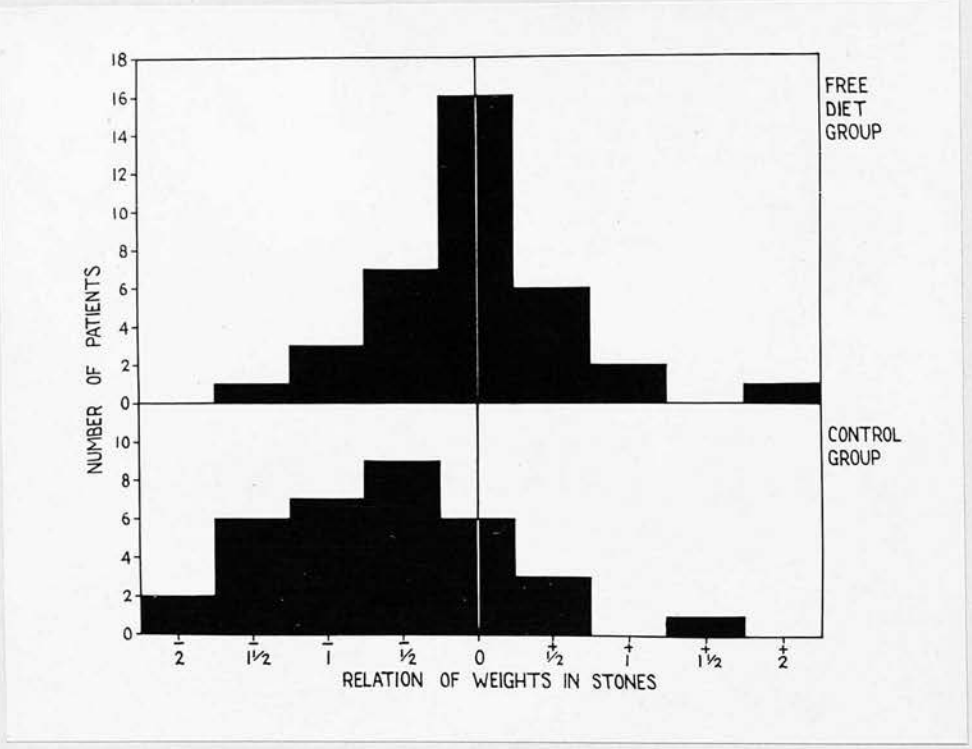


Relation of weight to correct weight - absolute.

Control Group - 40 patients

Precursorily obese - 16 patients
Precursorily normal - 11 patients
Precursorily thin - 7 patients
Unknown - 6 patients

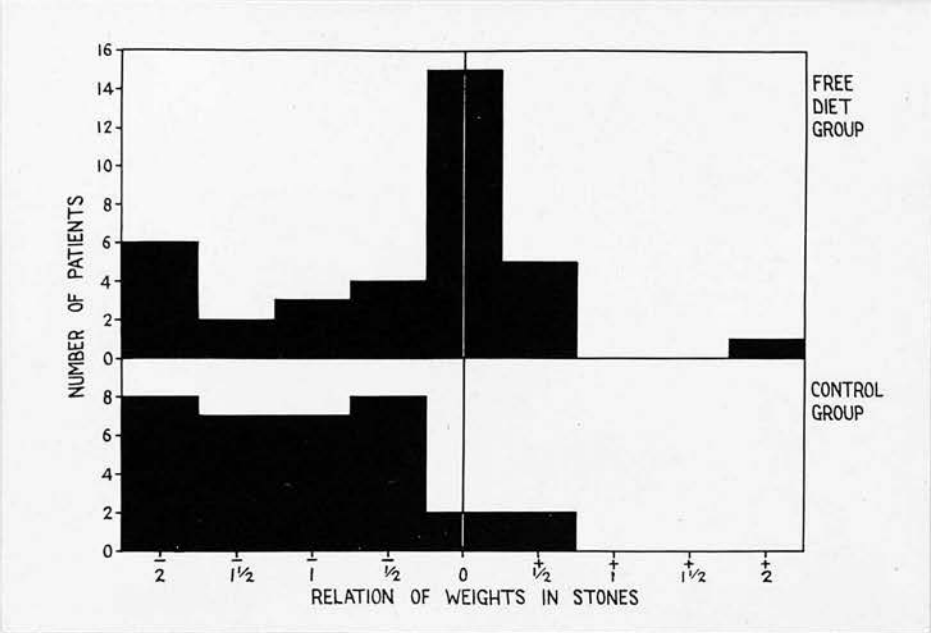
Graph 9.



Relation of weight to average weight, averaged to the nearest half stone.

Free Diet Group - 36 patients
Control Group - 34 patients

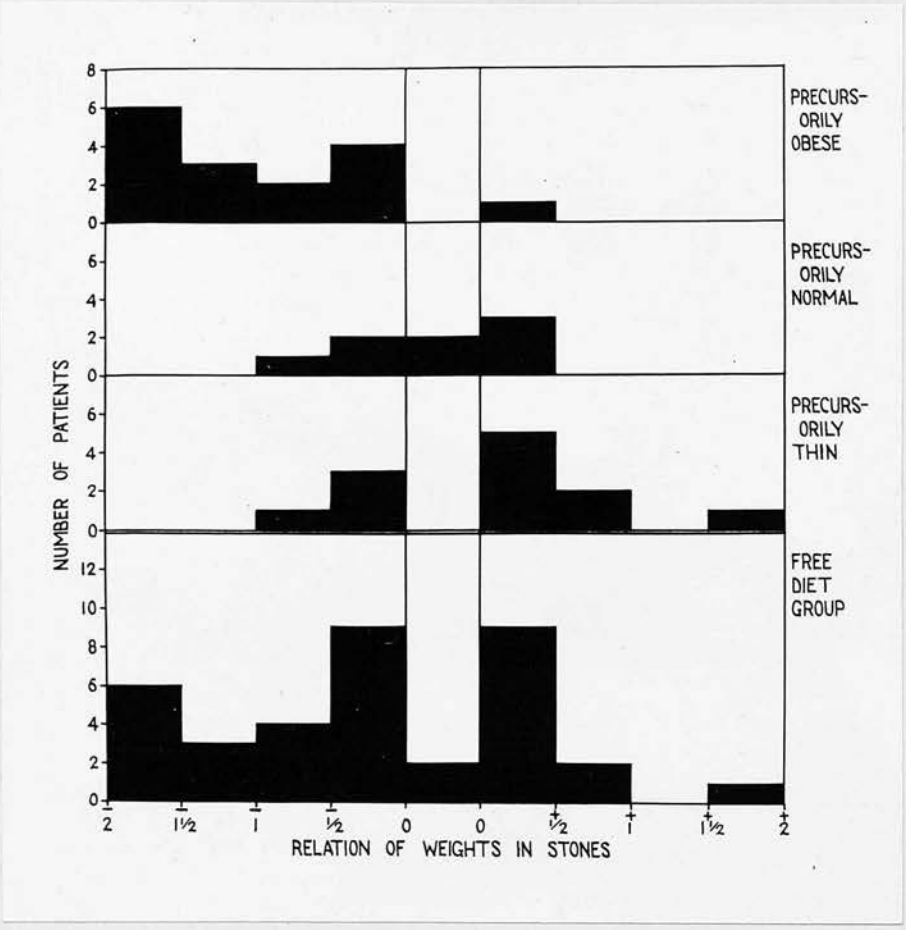
Graph 10.



Relation of weight to highest weight, averaged to the nearest half stone.

Free Diet Group - 36 patients
Control Group - 34 patients

Graph 11.

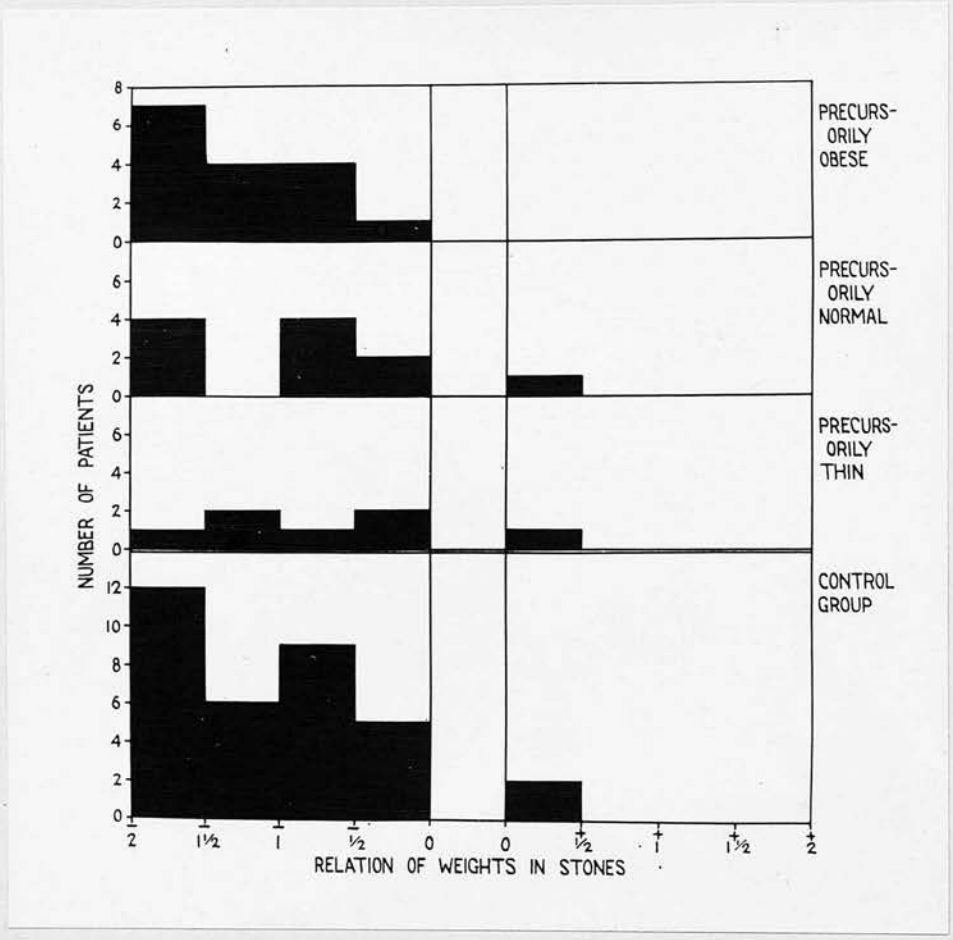


Relation of weight to highest weight - absolute.

Free Diet Group - 36 patients

Precursorily obese - 16 Patients
Precursorily normal - 8 patients
Precursorily thin - 12 patients

Graph 12.

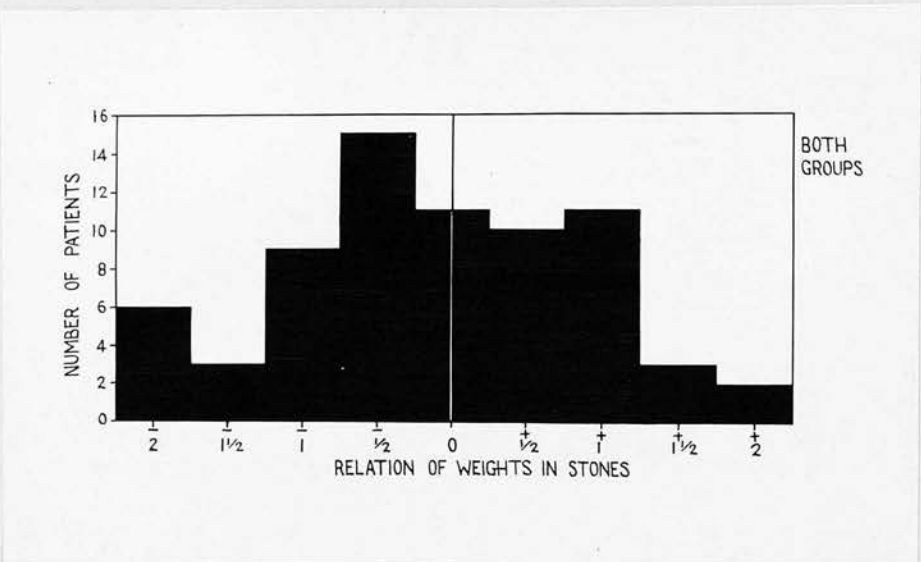


Relation of weight to highest weight - absolute

Control Group - 34 patients

Precursorily obese - 16 patients
Precursorily normal - 11 patients
Precursorily thin - 7 patients

Graph 13.



Relation of average weight to correct weight, averaged to the nearest half stone.

Both Groups - 70 patients.

Graph 6.

The free diet group are, on the whole, of normal weight and no marked tendency to obesity is seen.

The control group are also mostly of normal weight but a bigger proportion of them are thin than is the case with the free diet group.

Graph 7.

On a free diet the tendency shown, prior to the onset of diabetic symptoms, of obesity, normal weight or thinness, is maintained.

Graph 8.

On controlled diets the tendency to obesity is maintained but patients who were normal or thin tend to lose weight.

Graph 9.

On free diets, patients tend to regain their average weight of the past, while on controlled diets they tend to be thinner.

Graph 10.

Patients on free diets tend to regain the highest weight recorded prior to the onset of diabetic symptoms. The control group remain lighter in weight.

Graph 11.

On a free diet, the precursorily obese patient does not usually become as fat as he once was, the precursorily normal weight diabetic tends to regain his normal weight and the precursorily thin patient tends to become heavier than ever before.

Graph 12.

On a controlled diet, the precursorily obese patient definitely does not regain his highest weight, the precursorily normal weight diabetic tends to become thin, and the precursorily thin diabetic shows no tendency to become heavier as he does on a free diet.

Graph 13.

This graph is intended purely as a check upon the scale of "correct weights" used. The average weights of patients, before the onset of pre-diabetic obesity or before the loss of weight due to frank diabetes, and the correct weights are practically identical.

Summary.

Precursorily obese patients tend to remain obese even on controlled diets. The tendency is even more marked on free diets. The mechanism of this "food sparing phenomenon" is not understood.

Patients of precursorily normal weight regain that weight on free diets but tend to become thin if their calorie intake is restricted.

Precursorily thin patients tend to remain below the correct weight on free diets but, nevertheless, they often become heavier than they have ever been in the past. This is not evident where restricted diets are used and, indeed, on such a régime thin patients tend to become thinner than ever.

If a normal weight is the therapeutic aim, the precursorily obese should be given restricted diets even although actually underweight when they first come for treatment, whereas the precursorily normal or thin diabetics may be given diets of normal calorie value. Optimal results are not achieved in the latter two groups with subcaloric diets.

In the prescription of a diet for a new diabetic coming for treatment, his weight prior to the onset of frank diabetes should be a guiding factor.

As regards free diets, from the point of view of the possible development of obesity, they are best avoided in patients with a marked history of precursory overweight.

The results obtained in this survey suggest that the danger of inducing obesity does not exist in those who have never been overweight in the past, and, indeed, that such patients do better on normal calorie diets than on restricted diets.

16. ENERGY.

The energy of the patients of both groups for their everyday work was assessed according to their own subjective opinion. This introduced a possible error due to the psychological attitude of the patients but, as the important issue was what the patient thought about himself, the assessment was considered worth while.

Patients were graded I, II or III according to their answers to the questions given below.

1. "Do you feel as well as you did before becoming diabetic and able for as much physical work and exercise as ever before?"

   Patients answering 'yes' were placed in Grade I.

2. Those who replied 'no' were asked a further question -

   "Do you notice tiredness only occasionally when you are working particularly hard?"

   Patients answering 'yes' were placed in Grade II.

3. If this second answer was 'no', a final question was asked -

   "You mean that although you can manage a day's work you have noticed yourself easily tired on many occasions?"

   Patients answering 'yes' were placed in Grade III.

Grades I and II were satisfactory but Grade III was not.

The results are given in the following table:

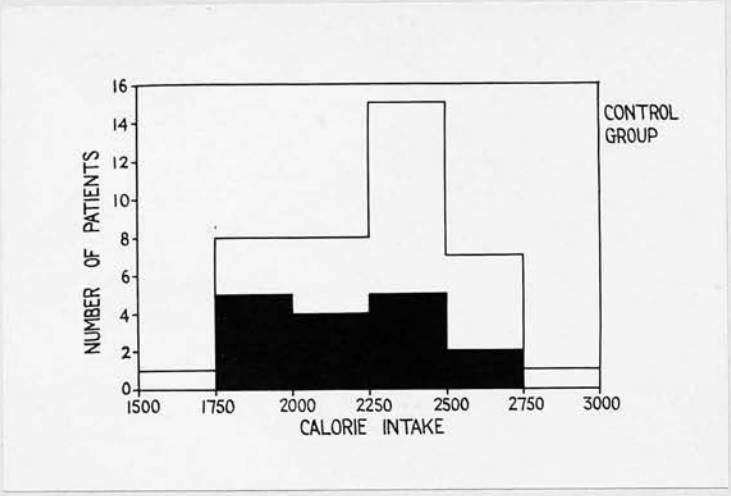
Group	Grade I Full energy	Grade II Practically full energy	Grade III Rather easily tired	Total
Free	37	8	2	47
Control	18	6	16	40

It is notable that nearly half of the control group were rather easily tired.

Tiredness in a diabetic may be due to inadequate calorie utilisation usually reflected in a subnormal weight. This may arise from the prescription of inadequate diets or from the more obvious cause of poor diabetic control. Low blood sugars, not far above the reactive level, may also lead to tiredness and finally the psychological attitude of the patient to his disability is vitally important.

The two free diet patients in grade III were tired, in one instance for psychological reasons, in a patient who has in the past been admitted to a mental institution for depression, and in the other case because of poor diabetic control, in a man of low mentality.

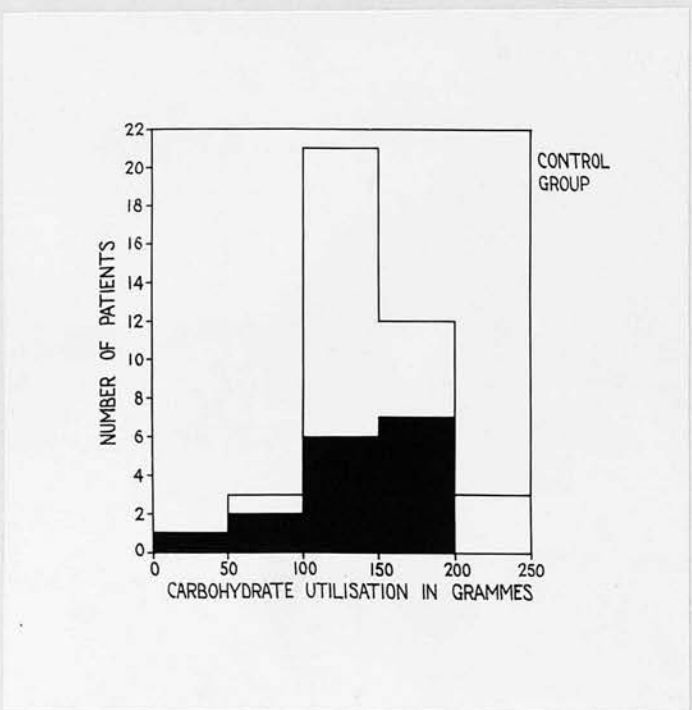
Graph 14.



Daily Calorie Intakes prescribed for Grade III patients of the Control Group compared with those of the Group as a whole.

Control Group - 40 patients - outlined
Grade III Group - 16 patients - in black

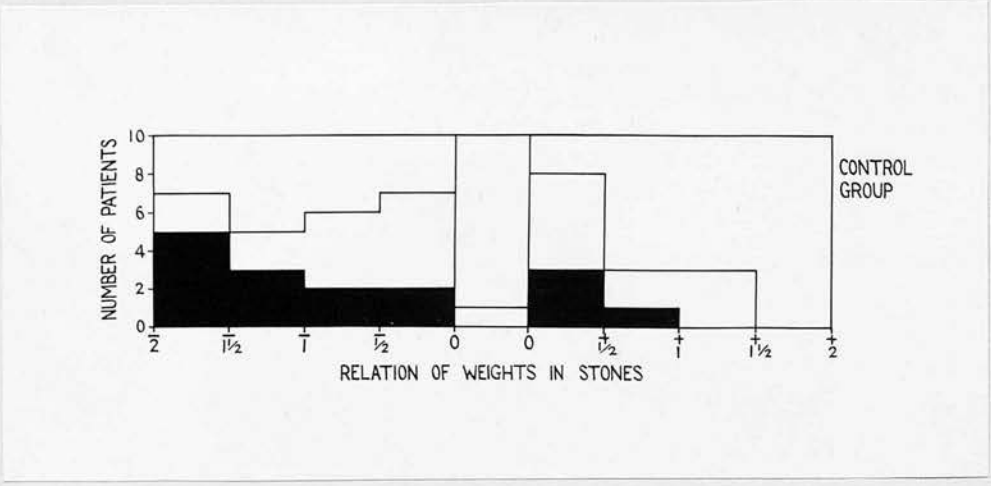
Graph 15.



Daily Carbohydrate Utilisations of the Grade III patients of the Control Group compared with those of the Group as a whole.

Control Group - 40 patients - outlined
Grade III Group - 16 patients - in black

Graph 16.



The relations of the weights to the correct weights of the Grade III patients of the Control Group compared with those of the Group as a whole.

Control Group - 40 patients - outlined
Grade III Group - 16 patients - in black

Graph 14.

The calorie intakes of the tired members of the control group were representative of the group as a whole.

Graph 15.

The carbohydrate utilisations of the tired members of the control group were also in no way different from those of the group as a whole.

Graph 16.

Twelve of the sixteen tired patients were below their correct weight. The remaining four, although still above the correct weight had been very obese in the past and had lost a great deal of weight.

Summary.

The calorie requirements of patients differ greatly and some, at least, of the dietetically controlled patients may be receiving diets too low for optimal energy. The excellent physical energy exhibited by the free diet group as a whole is striking.

17. URINE SUGAR LEVELS.

Apart from clinical findings, the degree of glycosuria is the commonest gauge of diabetic control used in practice.

Provided the renal threshold is within normal limits, freedom from glycosuria throughout the twenty-four hours means freedom from hyperglycaemia. The greater the glycosuria over a twenty-four hour period, the higher the blood sugars and the longer the duration of hyperglycaemia as a general rule.

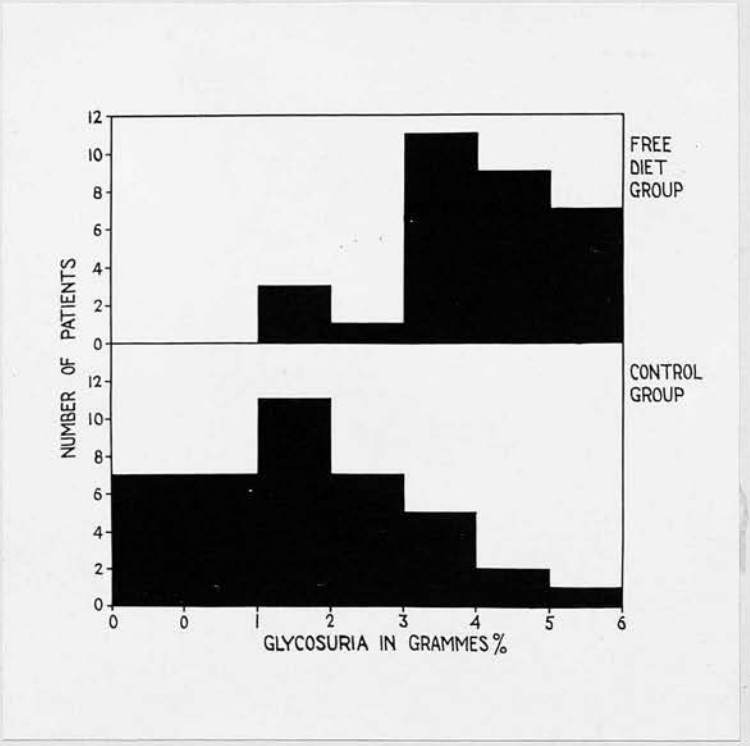
Thirty-one of the free diet group were co-operative in collecting accurate twenty-four hour samples of urine which were measured with a pint measure and specimens of which were brought for testing. The figures presented are an average of six twenty-four hour collections made consecutively, approximately once monthly, over a period from June 1948 to February 1949.

Nine patients brought collections made on Sunday and these showed poorer control than those from a working day. The remaining twenty-two patients were able to provide samples collected on working days. Meal charts were also brought covering the food eaten over the twenty-four hour period during which the collection was made.

The forty patients of the control group were asked to bring one twenty-four hour collection only and a corresponding meal chart.

From the data obtained results are charted of percentage glycosuria, twenty-four hour urine volume converted from pints to ml., twenty-four hour carbohydrate loss, twenty-four hour carbohydrate utilisation and the daily carbohydrate loss as a percentage of the carbohydrate intake.

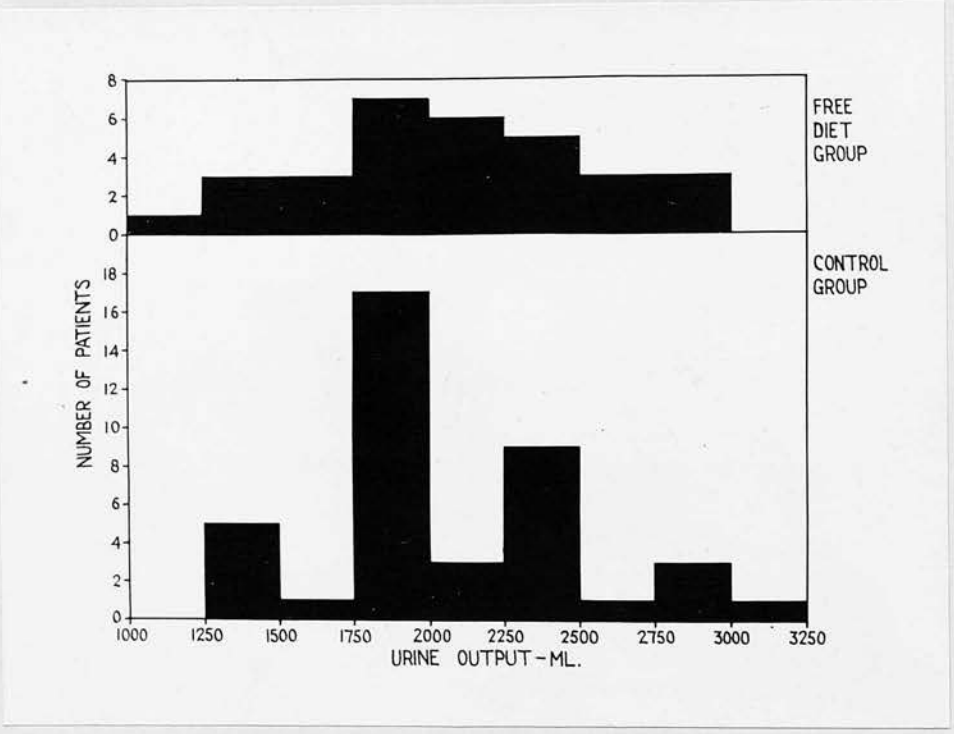
Graph 17.



Twenty-four hour percentage glycosuria.

Free Diet Group - 31 patients
Control Group - 40 patients

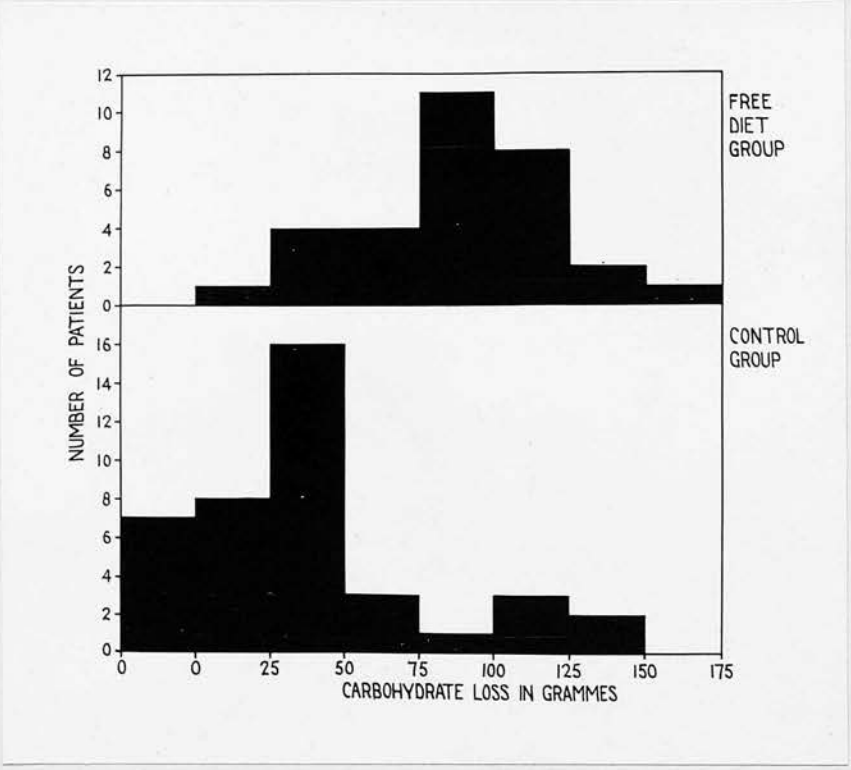
Graph 18.



Twenty-four hour output of urine.

Free Diet Group - 31 patients
Control Group - 40 patients

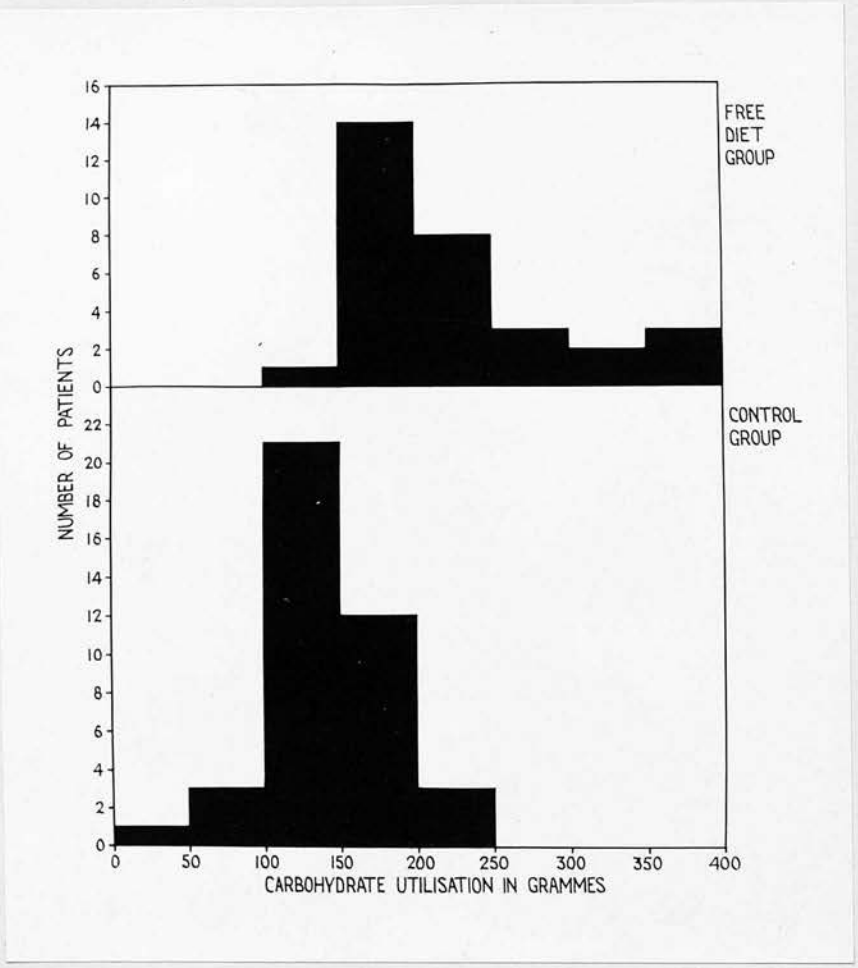
Graph 19.



Twenty-four hour carbohydrate loss.

Free Diet Group - 31 patients
Control Group - 40 patients

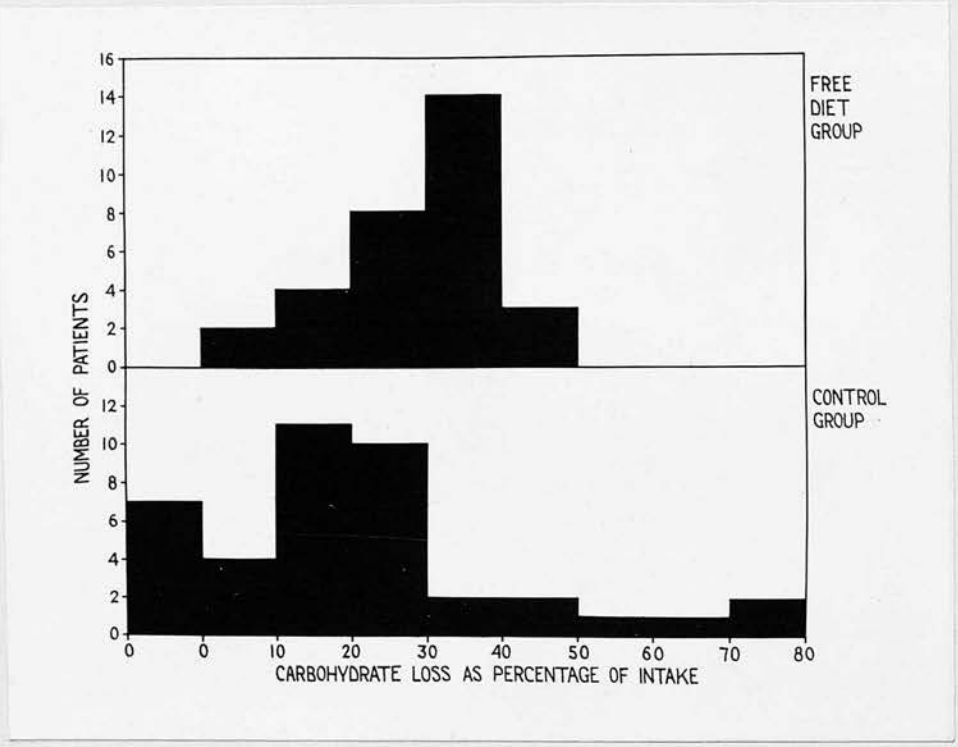
Graph 20.



Twenty-four hour carbohydrate utilisation.

Free Diet Group - 31 patients
Control Group - 40 patients

Graph 21.



Daily carbohydrate loss as a percentage of the carbohydrate intake.

Free Diet Group - 31 patients
Control Group - 40 patients

Graph 17.

The majority of free diet patients show a glycosuria of 3-6 grammes per cent in the twenty-four hour samples. Dietetically controlled patients have a lower sugar loss, usually between 0-3 grammes per cent.

Graph 18.

The twenty-four hour urine output is similar for both groups.

The absence of any significantly greater degree of polyuria in the free diet patients is notable.

Graph 19.

Carbohydrate loss in the twenty-four hours is greater in the free diet group, the peak incidence being 75-125 grammes compared to 25-50 grammes for the control group, i.e. the free diet group pass approximately 50-75 grammes more sugar in the twenty-four hours.

Graph 20.

Despite the heavy glycosuria, the free diet group, because of a higher carbohydrate intake which is more than compensatory, show a greater daily carbohydrate utilisation, namely, 150-250 grammes for the majority of patients compared with 100-200 grammes for controls.

The greater utilisation of carbohydrate by the free diet group no doubt accounts for their higher weights and excellent physical energy.

Graph 21.

The peak carbohydrate loss for the free diet group is 20%-40% of the intake compared with a loss of 10%-30% of the intake on controlled diets.

Summary.

The absolute twenty-four hour carbohydrate loss is greater in the free diet group than in the control group but this does not lead to any relatively increased polyuria.

Because of the unrestricted nature of free diets, the carbohydrate loss as a percentage of the intake is not so markedly higher and the actual utilisation of carbohydrate in the twenty-four hours is greater.

The results may be summarised very approximately as follows. The free diet patients eat about 125 grammes of carbohydrate daily more than the controls. They lose approximately 75 grammes more and the resultant carbohydrate utilisation is on the average 50 grammes greater in the twenty-four hours.

Clinical symptoms of thirst and polyuria are dependent upon the absolute carbohydrate loss. Patients of the free diet group with twenty-four hour carbohydrate losses of 125 grammes or less are entirely symptom free. In three patients losses are more than 125 grammes in twenty-four hours. Two of these patients brought collections from Sunday and, in the third, clinical control is borderline. Nocturia occurs two nights in the week.

Acetonuria, subnormal weight and tiredness are due to inadequate carbohydrate utilisation. Acetonuria must be avoided always if treatment is to be regarded as successful. The control group, however, show a higher incidence of subnormal weight and tiredness. To improve the carbohydrate utilisation of dietetically controlled patients of the non-obese type, it would be more practicable to raise their diets than to attempt reduction of their glycosuria by raising the insulin dosage alone, because of the danger of insulin reactions inherent in a method which attempts too fine a control of glycosuria.

18. BLOOD SUGAR LEVELS.

Of those remaining on a free diet in August 1948, twenty-five lived in Edinburgh and were willing to have blood sugars taken on a working day under home conditions.

Samples were withdrawn

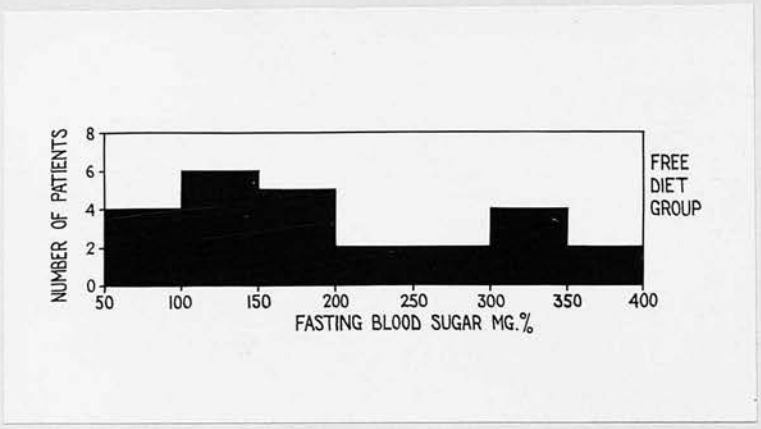
1. before lunch and then
2. before the evening meal and insulin injection. The patient went home for supper, then returned to sleep overnight in the ward and
3. a fasting blood sample was taken the following morning.

The day was picked at random during a period of clinically satisfactory control and freedom from infection. No blood sugar estimation was made within two hours of a meal.

It will be appreciated that the results provide only a rough guide to the degree of hyperglycaemia on free diets.

Thirty-four of the control group were willing to co-operate and samples were taken before lunch and before the evening meal from them. Fasting sugars were not included.

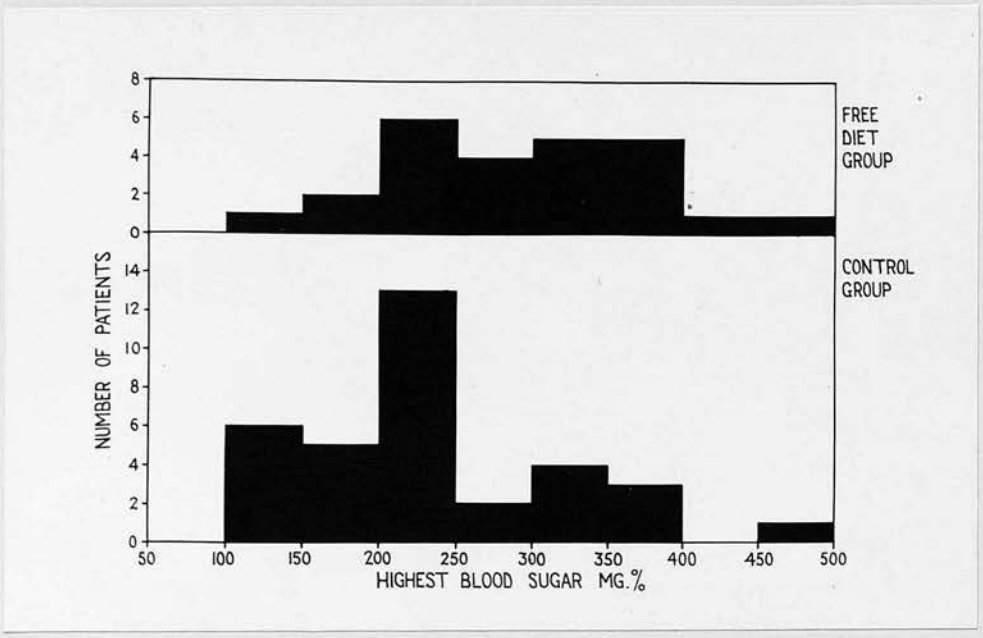
Graph 22.



Fasting Blood Sugar Levels.

Free Diet Group - 25 patients

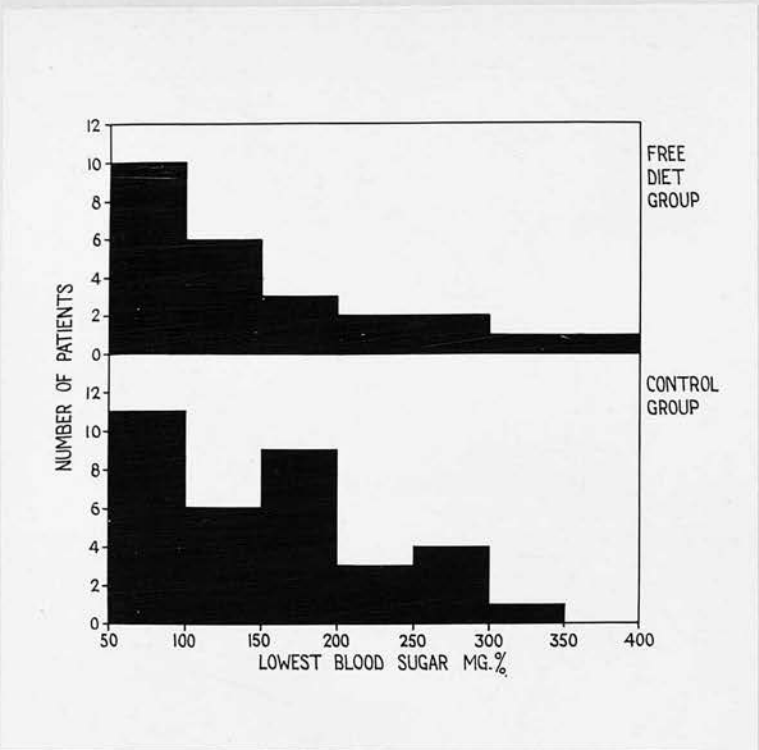
Graph 23.



Highest Blood Sugar Levels.

Free Diet Group - 25 patients
Control Group - 34 patients

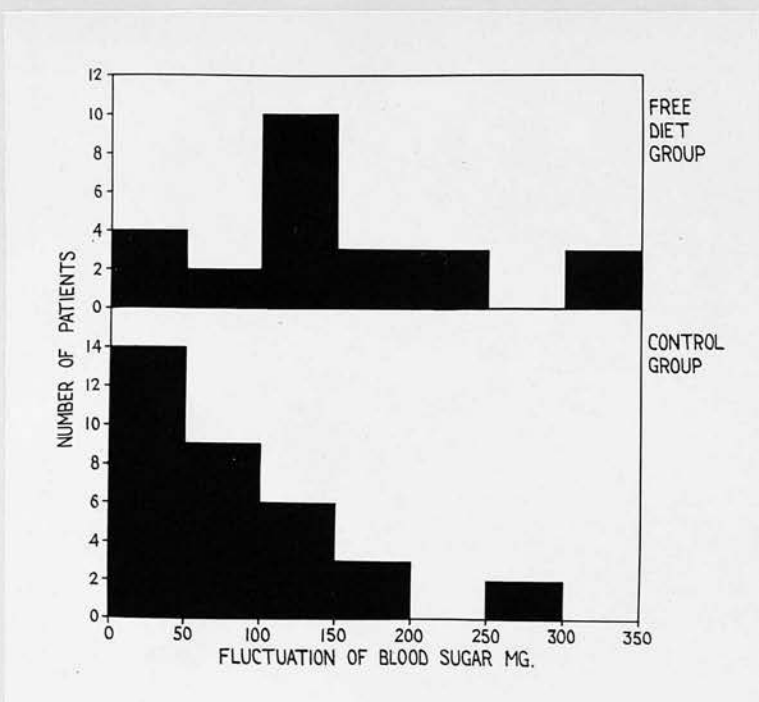
Graph 24.



Lowest Blood Sugar Levels.

Free Diet Group - 25 patients
Control Group - 34 patients

Graph 25.



Fluctuation of Blood Sugar Levels.

Free Diet Group - 25 patients
Control Group - 34 patients

Graph 22.

The fasting blood sugar levels for the patients studied on a free diet lie between 50 mg. % and 400 mg. %.

Graph 23.

The highest blood sugar levels recorded for both groups lie between 100 mg. % and 500 mg. %.

Hyperglycaemia above a level of 200 mg. % is present in 22 out of 25 free diet patients (88%) and in 23 out of 34 control patients (68%).

Hyperglycaemia of 350 mg. % or below is exhibited by 18 out of 25 free diet patients (72%). A level of 250 mg. % or below is the highest reached by 22 out of 34 control patients (65%).

Graph 24.

The lowest blood sugar levels for both groups are very similar.

16 out of 25 free diet patients show figures below 150 mg. % (64%).

17 out of 34 (50%) of the control group also exhibit levels below 150 mg. %.

From this result, there would appear to be no marked difference in insulin sensitivity between the two groups, even taking into account the fact that the free group on average take approximately 10 units

of insulin daily more than the control group.

Graph 25.

The fluctuation of blood sugar levels is undoubtedly less in the dietetically controlled patients studied.

If 100 mg. is taken as a normal fluctuation, 6 out of 25 (24%) of the free diet group comply, whereas that small variation is shown by 23 out of 34 (68%) of the control group.

Summary.

As already pointed out, the blood sugar levels quoted give only a very rough guide to the degree of hyperglycaemia shown by patients on free and controlled diets.

It is important to realise, however, that, even in a group of patients considered very co-operative from the dietetic clinic, blood sugars of over 200 mg. % were recorded before a meal in more than half of the patients and, as a corollary of the urine sugar results, the blood sugar rose above the renal threshold in at least three quarters of the dietetically controlled patients, some time in the twenty-four hours.

Hyperglycaemia is undoubtedly more frequent and more severe in patients on free diets. However, the choice of treatment lies, by no means, between a dietary régime readily attaining the ideal of normoglycaemia and a free method with attendant hyperglycaemia. The difference is purely one of degree and normoglycaemia cannot be assured on any method permitting good nutrition without grave danger of severe insulin reactions.

If, nevertheless, a strict control of glycosuria is insisted upon, a very much more rigid method must be enforced even with the co-operative members of the Dietetic Clinic.

19. INSULIN SENSITIVITY TESTS.

Himsworth (1936, 1939, 1940) reported the possible separation of diabetics into insulin-sensitive and insulin-insensitive types, by the use of an insulin-glucose test. He found no intermediate response to insulin.

The insulin-sensitive group were, on the whole, young, thin diabetics of acute onset, with normal blood pressure and vessels, subject to ketosis and insulin reactions and they responded well to high carbohydrate diets. He thought their diabetes might be due to a simple lack of pancreatic insulin.

The insulin-insensitive group were, in contrast, older, obese diabetics of slow onset, with hypertension and vascular disease, not subject to ketosis or insulin reactions and they responded poorly to high carbohydrate diets. Their diabetes, he thought, might have been due to the inhibition of insulin action.

MacBryde (1936) carried out tests using insulin alone without the simultaneous administration of oral glucose. He tried both subcutaneous and intravenous routes and found the results of both methods to correspond. He thought the separation of two groups to be possible. The insulin-sensitive group were young, thin and had normal blood pressures and

were liable to coma. The insulin-insensitive group were older, obese, hypertensive and not liable to coma. In contradiction of Himsworth's results, however, he found that the insulin-insensitive group did better on high carbohydrate diets.

Soskin and Levine (1938) agreed with MacBryde that the older insulin-insensitive patients reacted favourably to high carbohydrate diets. They thought that the high carbohydrate diets improved liver function with resultant amelioration of the diabetic state.

De Wesselow and Griffiths (1938), using Himsworth's insulin-glucose test, found no evidence for the two groups and observed that the type of response to insulin obtained was not related to age, weight or blood pressure readings.

Klatskin (1938), following MacBryde's method of a subcutaneous insulin sensitivity test, without oral glucose, found no division into two types and concluded that no significant relationship existed between the insulin sensitivity of diabetics and their clinical characteristics or their response to high carbohydrate diets.

Joslin (1947) also reported that there was no evidence in favour of insulin-sensitive and insulin-insensitive groups and that results did not correlate with clinical findings.

A clear differentiation of insulin-sensitive and insulin-insensitive types of diabetes would be of practical value in selecting patients for liberal or restricted forms of dietary treatment. According to Himsworth's view, one would expect insulin-sensitive diabetics to do best on liberal diets.

Insulin sensitivity tests were carried out on seventeen free diet patients and ten control patients from the Dietetic Department, in August 1948. Four of the latter patients were on dietary treatment alone and were not included in the control group of forty, which are used for comparison throughout the survey.

The standard insulin sensitivity test was employed, namely 0.1 unit of soluble insulin per kilogram of body weight intravenously, after the withdrawal of a sample of fasting blood.

This test was criticised by Himsworth (1936) who preferred a glucose-insulin test, but, nevertheless, it was felt that any clear-cut differences in sensitivity would manifest themselves with the cruder test and that the alimentary absorption rate of an oral dose of glucose would introduce a further possible variable.

Patients were clinically fit at the time of the test and free from the action of subcutaneously

administered insulin. Their last meal was at 8-30 p.m. the previous evening and the last insulin injection, in the form of soluble only, amounting to half of the total usual daily requirement, was given the previous morning.

No patient smoked during the test and strict rest in bed was maintained. Insulin of strength 20 units per c.c. was administered from a tuberculin syringe kept specially for the purpose. Blood sugars were taken ½ hour, 1 hour and 2 hours after the administration of insulin.

Results.

Complete details of individual results are given in the appendix. The deductions from these are summarised in the following tables.

Distribution of Patients according to percentage fall of blood sugar.

Total Number	Percentage fall of blood sugar						
	21-30	31-40	41-50	51-60	61-70	71-80	81-90
27	3	6	8	5	1	2	2

Comment.

The twenty-seven patients show a wide variation of sensitivity to insulin. No clear-cut differentiation into insulin-sensitive and insulin-insensitive types is possible.

Percentage fall of blood sugar
in relation to age.

Total Number	Age Years	Percentage fall of blood sugar						
		21-30	31-40	41-50	51-60	61-70	71-80	81-90
3	11-20	1	0	1	1	0	0	0
2	21-30	0	0	2	0	0	0	0
12	31-40	2	4	1	3	0	0	2
4	41-50	0	0	3	0	0	1	0
2	51-60	0	1	0	1	0	0	0
4	61-70	0	1	1	0	1	1	0
27		3	6	8	5	1	2	2

Comment.

There is no obvious relation between age and the degree of sensitivity to insulin exhibited by the patients.

Percentage fall of blood sugar
in relation to dietary carbohydrate.

Total Number	Diet Ranges in Grammes of Carbo-hydrate	Percentage fall of blood sugar						
		21-30	31-40	41-50	51-60	61-70	71-80	81-90
1	51-150	0	0	0	0	1	0	0
13	151-250	0	3	4	3	0	2	1
6	251-350	2	2	1	1	0	0	0
4	351-450	0	1	1	1	0	0	1
3	451-550	1	0	2	0	0	0	0
27		3	6	8	5	1	2	2

Comment.

Patients on diets below 250 grammes of carbohydrate daily on the whole show a greater sensitivity to insulin than those on higher diets.

Percentage fall of blood sugar
in relation to percentage glycosuria.

Total Number	24 hour Glycosuria in Grammes %	Percentage fall of blood sugar						
		21-30	31-40	41-50	51-60	61-70	71-80	81-90
4	0	0	0	0	2	0	1	1
2	0.1-1	0	0	0	0	1	1	0
4	1.1-2	0	1	1	1	0	0	1
1	2.1-3	0	1	0	0	0	0	0
6	3.1-4	2	2	1	1	0	0	0
4	4.1-5	0	0	4	0	0	0	0
5	5.1-6	1	2	2	0	0	0	0
1	6.1-7	0	0	0	1	0	0	0
27		3	6	8	5	1	2	2

Comment.

Patients with minimal glycosuria are undoubtedly more sensitive to insulin than those with considerable glycosuria.

Summary.

Insulin sensitivity tests were found to be of no practical value in differentiating diabetics with a view to deciding the optimal diets to employ. There was no apparent relationship between insulin sensitivity and age. Patients on lower carbohydrate intakes and well controlled from the point of view of glycosuria were, in most instances, more sensitive to insulin than the others.

20. BLOOD SUGAR CURVES.

Consecutive blood sugar curves were done on eighteen patients being treated on a free diet. In each case half the total daily insulin requirement in the form of soluble insulin had been given the previous morning and half the previous evening. No food had been eaten since 8-30 p.m. the previous evening and the test was carried out at 9 a.m. under fasting conditions. Patients were kept at rest in bed throughout and no smoking was allowed. Venous blood was estimated.

The fasting levels under such conditions are of little value but the level one hour after the ingestion of 50 grammes of glucose in 180 ml. of water, considered along with the level after two hours, gives an indication of the severity of the diabetic curve. Where the regulatory mechanism is capable of response, the level after two hours is lower than that after one hour, and the degree of rebound gives an indication of the changing severity of the diabetic curve.

These tests were begun near the commencement of treatment on a free diet and, by repetitive tests, it was hoped to obtain an accurate gauge of the severity of the diabetic state and the response to high diets.

The complete data is given in the appendix.

Results.

	Initials	Assessment of the diabetic state from the degree of rebound. Better - Same - Worse	Assessment of the diabetic state from the daily insulin requirement. Better - Same - Worse	Correspondence of Results. Yes or No
1	T.E.	Worse	Worse	Yes
2	D.P.	Better	Same	No
3	R.Gr.	Better	Same	No
4	B.R.	Worse	Worse	Yes
5	J.Di.	Worse	Worse	Yes
6	L.H.	Worse	Worse	Yes
7	R.McW.	Worse	Same	No
8	J.McW.	Worse	Same	No
9	E.H.	Better	Worse	No
10	J.McWm.	Worse	Worse	Yes
11	H.S.	Worse	Worse	Yes
12	R.M.	Worse	Worse	Yes
13	J.L.	Better	Worse	No
14	G.S.	Better	Better	Yes
15	J.F.	Worse	Worse	Yes
16	A.C.	.Worse	Same	No
17	J.J.	Worse	Same	No
18	D.C.	Worse	Worse	Yes

Comment.

In the cases of D.P., R.Gr., E.H. and J.L. the better rebound occurred following a greater degree of hyperglycaemia at one hour from the ingestion of glucose.

In the cases of R.McW., J.McW., A.C. and J.J. the poorer rebound occurred following a lesser degree of hyperglycaemia at one hour.

It is thus clear how very difficult it is to assess the changing severity of the diabetic state by carrying out repetitive blood sugar curves. The comparative insulin dosage is not a true index of severity either, e.g. J.L. requires more insulin than he did three years ago, but this fact must be considered in relation to his growth between the ages of nine and twelve.

In conclusion, blood sugar curves were not found to add any significant weight to the assessment of the severity of the diabetic state over the years of treatment. The comparative insulin requirement is the best guide available but it is by no means more than a rough indication and must be considered along with clinical findings such as the changing weight of the patient and the degree of diabetic control resultant upon the insulin given.

The results of the assessment of the diabetic state, based upon the daily insulin requirements, are considered for the whole free diet group in the next section.

21. COMPARATIVE INSULIN REQUIREMENTS.

The dose of insulin required in March 1949, or at the time of the change-over to a controlled diet in the case of the eleven failures, is compared with the dose taken three months after the patient was first stabilised on a free diet.

The recent dose of insulin is expressed as a percentage of the original requirement with a plus or minus sign prefixed. All those now taking 20% or more insulin in excess of the original requirement are classified as "requiring more insulin". Those taking 20% less insulin than the original requirement are classified as "requiring less insulin". Others are classified as "the same".

For forty-seven uncomplicated diabetics who have been carefully followed up on a free diet, results are as follows.

Requiring more insulin	The same	Requiring less insulin
18	25	4

Five out of seven of the juvenile group, whose treatment was begun before the age of sixteen, require more insulin, which is to be expected with children of the puberty period.

Excluding the seven juvenile diabetics, one may consider the forty adults whose results are as follows.

Requiring more insulin	The same	Requiring less insulin
13	23	4

The following results entail a consideration of the insulin requirements in the light of the duration of free diet treatment, for the adult group.

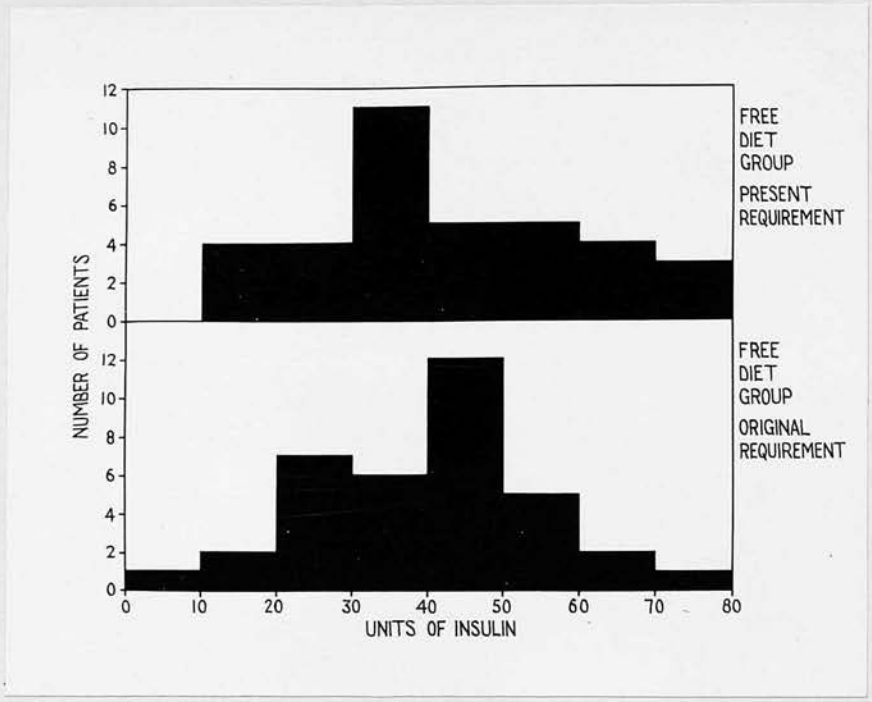
Duration of free diet treatment. Years	Total number of Patients	Number requiring more insulin	Proportion requiring more insulin (approx.)
$0-1\frac{6}{12}$	8	1	$\frac{1}{8}$
$1\frac{7}{12}-2\frac{6}{12}$	20	5	$\frac{1}{4}$
$2\frac{7}{12}-3\frac{6}{12}$	12	7	$\frac{1}{2}$

The insulin requirements have tended to rise with the duration of free diet treatment. It is uncertain at present whether, as the years go on, more patients will have to be changed to restricted diets because of ever increasing insulin requirements or whether the dose will become static once a stage of

"absolute" diabetes is reached, as forecast by Lawrence (1944) for diabetics receiving controlled diets.

The insulin requirements of the thirty-six patients remaining on free diets in March 1949 will now be considered in comparison with their original requirements and the requirements of the control group.

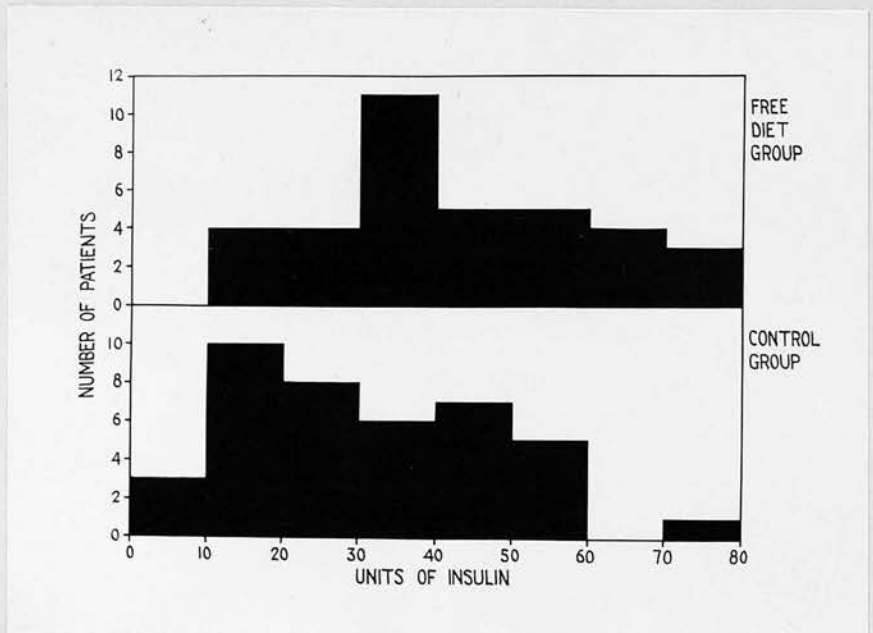
Graph 26.



Present daily requirements of insulin compared with the requirements when stabilisation was originally effected.

Free Diet Group - 36 patients

Graph 27.



Present daily requirements of insulin of the Free Diet Group compared with those of the Control Group.

Free Diet Group - 36 patients
Control Group - 40 patients

Graph 26.

The thirty-six uncomplicated diabetics remaining on free diets show no overall change in their requirements of insulin since the time when stabilisation was first effected.

Graph 27.

The daily insulin requirements of the free diet group of thirty-six patients are seen to be approximately 10 units daily in excess of those of the control group.

Summary.

Insulin requirements of the free diet group as a whole have tended to rise during treatment. Many patients, however, indeed more than half of the adult group, take virtually the same dose of insulin daily as at the commencement of free diet treatment. A longer follow-up will be required to see whether, because of a gradual increase in insulin dosage, more patients will have to be changed to weighed diets.

22. FAILURES.

Eleven patients out of the total of fifty-five who were given free diets had to be changed to a restricted or regulated food intake. Details are given in the following table.

	Initials	Age at onset of free diet treatment	Daily insulin at time of change to a strict diet	Reasons for change from free diet to strict diet
		Years	Units	
1	B.R.	14	76	Severity of diabetes
2	L.H.	14	88	Severity of diabetes
3	A.A.	54	16	Obesity - Pruritus
4	J.L.	9	42	Reactions frequent
5	J.F.	33	100	Severity of diabetes
6	A.Mc.	21	84	Severity of diabetes
7	A.N.	55	48	Reactions frequent
8	J.Pa.	16	92	Severity of diabetes - Pruritus
9	C.Wa.	16	84	Severity of diabetes
10	D.C.	16	80	Severity of diabetes
11	H.G.	28	48	Obesity - Pruritus

Seven of the failures on a free diet were severe diabetics who could not be controlled on eighty units of insulin per day. It was not considered justifiable

to raise their insulin further merely to maintain a free diet régime. Five of the seven were passing through the years of puberty, two were adults.

Frequent reactions on a dose of insulin adequate for diabetic control made it essential to regulate the diets of two patients.

The two other patients were changed to subcaloric diets because of a tendency to obesity and pruritus vulvae.

Summary.

Caution in the use of free diets for young people of puberty age and for adults with a marked history of precursory obesity would eliminate most failures. However, some patients, whose diabetes might become too severe for a free diet or who might experience many reactions unless given a regulated food intake, would still appear for treatment and there is no method of distinguishing them except by trial and error.

23. SUMMARY OF THE RESULTS OF THE PRESENT INVESTIGATION.

The short-term results of a liberal method of dieting in the treatment of fifty-five diabetics are compared with those of the method in use in the Dietetic Department in which all of the dietary constituents are weighed.

The liberal method under investigation allowed a free choice of foodstuffs except for the avoidance of sugar, jam and sweets. Patients usually adopted a system of three main meals with a snack in the morning and before going to bed at night. Assessment of the diets chosen revealed them to be essentially normal in the proportions of carbohydrate, fat and protein and in the total calorie value. The carbohydrate intake was considerably higher than that usually prescribed in diabetic diets and a wider variation of intake was seen from patient to patient than is allowed for in the weighed diet method at present in use. Diets remained remarkably constant during the week but varied at weekends to suit differences in exercise on Saturdays and Sundays.

The clinical control of the free diet group was maintained at a satisfactory level by the use of insulin which was given twice daily in the majority

of patients in a combination designed to allow maximal control with minimal risk of reactions. As a result, reactions were infrequent. The total daily dose of insulin required was slightly greater on the whole than that used for the dietetically controlled group.

In every patient remaining on a free diet freedom from thirst, pruritus and acetonuria was achieved. A certain degree of polyuria was permitted so long as patients did not complain of frequency of micturition or nocturia. On the whole, those receiving a free diet exhibited a greater degree of physical energy than the control group.

On free diets nutrition was good. Weight studies suggested that the danger of the development of obesity during treatment existed only in those with a marked history of overweight before the onset of diabetic symptoms. Diabetics who had been of normal weight or thin in the past regained their usual weight on free diets in contrast to those on restricted diets who were frequently underweight. Precursorily obese diabetics do better on restricted diets but other adult diabetics requiring insulin might, with benefit, be allowed diets of normal calorie value.

The growth and development of the small group

of juvenile diabetics treated on liberal diets were very satisfactory. Seven out of eight women who were hoping to have children became pregnant.

Two patients developed diabetic coma during treatment on free diets. The incidence of pyogenic complications as a whole was not high but, perhaps because of two injections of insulin daily, four injection abscesses occurred among those on free diets. As regards degenerative complications, four patients were found to have diabetic retinitis and one an early senile cataract.

Urine sugar results showed a greater degree of glycosuria among free diet patients than among those on controlled diets. Nevertheless, the urine outputs were similar for both groups and, despite the greater sugar loss, carbohydrate utilisation was greater in the free diet group.

Hyperglycaemia was present in those receiving controlled diets as well as in those receiving free diets and it occurred with greater frequency and severity in the latter.

A tendency to a rise in insulin requirements occurred during free diet treatment and whether this will continue in the future, making a change to controlled diets necessary, is uncertain. An initial worsening of the diabetic state is recognised over

the first years of treatment even on a strict régime.

The free diet method was found to be unsuitable for five young people of puberty age and for two adults with a marked history of precursory overweight. The puberty age group and precursorily obese adults would not be given free diets in the future. It became impossible to control two adults on free diets because of thirst and polyuria despite large doses of insulin and one adult and a little boy had to be given regulated diets because of frequent insulin reactions. The latter four failures on a free régime could not have been predicted. Altogether forty-four of the original fifty-five patients given free diets, as defined, are still being treated by that method, and their clinical control is satisfactory.

Chapter VIII.

DIABETES MELLITUS IN THE YOUNG PATIENT.

Juvenile diabetes presents a difficult therapeutic problem. It is usually of greater severity than adult diabetes and ketosis and coma are more frequent.

There is suggestive evidence that a child, who has just developed diabetes, is physically ahead of his years and that this is due to pituitary overactivity.

Overheight at the onset of the disease has been reported by Boyd and Nelson (1928), Spencer (1928), Rabinowitch and Bazin (1929), and Priscilla White (1947). The latter mentioned also the occurrence of bone development eighteen months in advance of the chronological age, dental development twelve months in advance, a basal metabolic rate averaging + 12%, and an earlier onset of puberty in girls and boys with diabetes than in normal children. Brown and Thompson (1940), however, noted no consistent or peculiar deviations from the average height in diabetic children, at the onset of the disease.

If pituitary overactivity is the basic disorder in juvenile diabetes it would appear rational to

employ a therapeutic procedure to reduce the over-activity. It is believed that undernutrition inhibits anterior pituitary action. However, it is important to realise that the anterior pituitary gland acts as a functional whole. Excessive depression of its diabetogenic action by low calorie diets results in depression of growth and sexual function as well.

That undernutrition in normal children results in poor growth is recognised by many writers including Harris (1947). Delay in the onset of puberty in girls, in the working-class commune of Belgium, due to the dietary restrictions associated with the German occupation, was described by Ellis (1945). Talbot and Sobel (1947) suggested the possibility that "a simple deficit or excess of calories may tend to modify pituitary function and hence the rate of growth and maturation".

The undernutrition treatment of diabetes appears, in the past, to have led to poor growth. Before the introduction of insulin, growth in diabetic children was a rarity (Joslin, Root and White, 1925). But, even since insulin, stunting has been reported in the literature by Spencer (1928), Rabinowitch and Bazin (1929), John (1935), McGavin et al. (1940) and Fischer et al. (1942). Priscilla White (1947)

encountered a higher incidence of shortness in diabetics than in normal children and in many cases there were associated subnormal levels of sex hormone excretion. She believed that the frequent concurrence of obesity pointed to an endocrine rather than a nutritional cause for stunting of growth.

In contrast, Boyd and Kantrow (1938) concluded that diabetes did not lead to retardation of growth so long as ample food and insulin were given. This opinion was also held by Greenblatt and Nieburgs (1948). Jackson and McIntosh (1945) advocated normal calorie diets for diabetic children. Using such diets, Jackson and Kelly (1946) reported a definite correlation between satisfactory increments in height and weight and the development of sexual maturity. Well controlled diabetics grew well and poorly controlled children grew very little. In some instances, exceptionally well controlled children showed accelerated growth curves. Lichtenstein (1938), using free diets, achieved normal growth and development in all his children except in a very few isolated cases.

The present investigation included seven young, uncomplicated diabetics of sixteen years or under at the commencement of treatment. Information regarding

their growth on liberal diets is given in the following table.

	Initials	Age at onset of Free Diet Treatment	Height at onset of Treatment	Normal Height at age of onset	Age in 1949	Height in 1949	Normal Height for age in 1949	Total Increment in height	Normal Increment in height for same period
		Years	Inches	Inches	Years	Inches	Inches	Inches	Inches
1	B.R.	14	59	60.7	17	67	65.4	8.0	4.7
2	L.H.	14	62	60.7	17	69	65.4	7.0	4.7
3	J.L.	9	52	51.2	12	58	57.1	6.0	5.9
4	E.Ber.	16	61.5	63.2	18	62	64.0	0.5	0.8
5	J.Pa.	16	66.5	63.2	18	67	64.0	0.5	0.8
6	C.Wa.	16	62	63.2	17	62.5	63.9	0.5	0.7
7	D.C.	15	64.5	62.4	16	66	64.7	1.5	2.3

The figures for normal heights according to age are taken from Engelbach's table given on page 290 of "Endocrine Disorders in Childhood and Adolescence" by Le Marquand and Tozer, London, 1943, page 290.

At the onset of treatment on a free diet, four children were above the average height and three below. In 1949, five are above the average height and two below. All the young people are within the normal height range.

The two patients below average height are girls

who were sixteen at the onset of treatment. In them and in the other girl of sixteen, growth had virtually stopped before the commencement of treatment. In all three, menstruation was already well established.

The four boys, however, all started free diet treatment in the growing period. Two show accelerated growth curves, one a normal increment and one has grown rather less than the expected increase. All four boys in 1949 are, nevertheless, above average height.

The story of J.L. illustrates the value of a liberal method of treatment in one particular child. He became diabetic at the age of seven years shortly after the death of his mother. His father, a labourer, was left with four boys to bring up, two older and one younger than J.L. The adherence to a weighed diet was impossible under such circumstances and for nearly two years he was looked after in a country branch of the Royal Hospital for Sick Children where he received a careful standard diet and 26 units of zinc protamine insulin daily. He kept very well except for occasional insulin reactions. In the two years, however, he grew only two inches, and his schooling was upset.

Thereafter he was sent to the Royal Infirmary where, for social reasons, he was given a free diet

and included in the present survey. His father has taken very good care of the boy and ensured regular injections of insulin. For three years he has not missed a day off school and growth has been entirely satisfactory. He still showed a tendency to reactions, however, until recently when his diet was regulated by a simple method, followed by the child himself, which did not involve the use of scales. There was no dietary restriction but merely a regulation of the carbohydrate intake at each meal, a method which has cut out insulin reactions entirely since its adoption.

Entirely free diets, except for sugar, jam and sweets, have not proved satisfactory in the treatment of the group of young diabetics and, in six out of the seven, some conscious regulation of the diet has become necessary.

J.L. was given a regulated diet because of insulin reactions; all of the others, except E.Ber., were changed to slightly restricted diets because of the impossibility of attaining clinical control on free diets, using eighty units of insulin daily. It was considered unjustifiable to raise the dose of insulin above eighty units per day purely for the sake of continuing the free diet method.

Lichtenstein (1945) found no difficulty in

treating children even during the years of puberty on free diets, employing up to 120 units of insulin daily.

The diets prescribed for the five young people of puberty age in the present series were unrestricted in protein and fat, but the carbohydrate content was reduced to approximately 250 to 300 grammes and the intake was kept virtually constant from day to day by a simple method not involving the weighing of foods. Such diets were considerably higher than those often prescribed in dietetic clinics.

To obtain full growth and development, large doses of insulin are necessary. Lichtenstein (1945) advocates up to 120 units daily; Payne (1947) prescribes 80-120 units for diabetic control during the period of puberty.

The daily insulin requirements for the seven young diabetics of this series are given below, for 1949, on the diets just outlined.

	Initials	Daily Insulin - Units
1	B.R.	76
2	L.H.	88
3	J.L.	42
4	E.Ber.	48
5	J.Pa.	92
6	C.Wa.	84
7	D.C.	80

In the treatment of juvenile diabetes a careful course must be steered between overfeeding and the symptoms of thirst and polyuria, and underfeeding, with consequent stunting of growth and delayed sexual development. The experience gained from this very small group of children suggests that free diets are not suitable, but good results can be obtained by simple unweighed diets of up to 300 grammes of carbohydrate daily with normal proportions of protein and fat. Such diets retain the full growth and maturation propensities of the anterior pituitary gland.

Diabetic coma is commoner in the young person than in the adult. The causes of diabetic coma quoted by Root and Marble (1947) are:-

(i) Too much food.
(ii) Too little insulin.
(iii) The presence of infection.

It is notable, however, that Sprague (1947) purposely omitted dietary indiscretion from his list of causes of diabetic coma.

Mirsky (1942) believed that hyperglycaemia was not dangerous from the point of view of the development of ketosis, but that a diminution of liver glycogen due to inadequate carbohydrate utilisation was the crucial factor. This, he thought, resulted

in an accelerated rate of acetone-body formation in the liver, and consequent ketosis. Himsworth (1949) put forward the view that hyperglycaemia acted as an aid to glycogen storage.

Payne (1938) described the onset of coma in diabetic children as closely resembling the cyclical vomiting of non-diabetics. It is the experience in Ward 21 that diabetic coma in children has appeared for no obvious cause and it could not be traced to any dietary indiscretion, omission of insulin or infection. Despite high diets, none of the seven young diabetics in this series developed diabetic coma during treatment.

A simple estimated diet lying between the extremes of undernutrition and free régimes has produced good clinical results to date. The diets employed were considerably higher than those frequently prescribed in dietetic clinics and, with appropriate large doses of insulin, the growth of the children has been excellent.

Chapter IX.

IS HYPERGLYCAEMIA HARMFUL?

INTRODUCTION.

In the non-obese adult diabetic requiring insulin, only the strictest régime of treatment can approach the ideal of perpetual normoglycaemia. Not only must the diets be restricted, but meals and insulin injections must be carefully timed and control imposed upon exercise because of the risk of insulin reactions inherent in the method. It is, moreover, impossible for any physician to claim that his patient is always sugar-free. Twenty-four hour urine collections are the only guide and these cannot be obtained with sufficient frequency.

Many errors creep into the weighed diet method. Scales are often fragile and inaccurate. The patience with which a slice of bread is weighed must vary greatly from patient to patient and the slightest excess on each slice may readily lead to an extra 100 grammes of carbohydrate in the day's total. On rigid methods the defaulting rate is high and regular attendance at a diabetic clinic is in itself very important. On the other hand, the more co-operative the patient in adhering to a weighed diet, the greater the liability to neurosis.

Some clinics have given up the prescription of diets of accurate calorie value involving the weighing

of the carbohydrate, protein and fat contents of meals and now advocate the weighing of carbohydrate only. Such clinics no longer adhere to the principle of undernutrition. Other clinics are satisfied with "estimated" diets where the portions of carbohydrate are assessed according to size by the use of models. The individual is certain to be generous with himself on such a régime and the outcome cannot be far removed from that of the virtually free method of dieting studied in this investigation. These simpler methods have the great advantage of allowing the important things to be clearly grasped by patients who are in no danger of becoming obsessed by complicated food exchanges or horrified at the appearance of a positive Fehling's test.

With any system, except that involving the careful weighing of all the dietary constituents, hyperglycaemia is likely to occur and, the more liberal the method, the greater the liability to hyperglycaemia.

There are three major arguments against the use of methods incurring the risk of hyperglycaemia, namely -

1. Hyperglycaemia may obviate the possibility of good clinical control.
2. Hyperglycaemia may damage the pancreas.
3. Hyperglycaemia may lead, after a latent period, to the development of degenerative complications.

1. HYPERGLYCAEMIA AND CLINICAL CONTROL.

It is important to distinguish the hyperglycaemia, which is of endogenous origin due to the break-down of glycogen stores, and which is accompanied by poor carbohydrate utilisation and ketosis, from the hyperglycaemia of exogenous origin, resulting from an abundant carbohydrate intake, with which, however, carbohydrate utilisation is adequate and there is no associated ketosis. This distinction is rarely made in the literature and harmful effects, in patients who have defaulted from the dietetic régimes imposed and have not attended regularly at a clinic, are blamed on hyperglycaemia rather than on poor control of the diabetic state.

Controversy exists only as to the harmfulness of exogenous hyperglycaemia or, as it is more often called, hyperglycaemia per se, when the associated clinical control is satisfactory.

Hyperglycaemia, in the presence of a normal renal threshold, leads to glycosuria. On glycosuria depend the clinical symptoms of thirst, polyuria and pruritus. Exponents of the free diet method claim that the degree of glycosuria obtained is insufficient to cause symptoms. The opponents of liberal régimes believe symptoms to be inevitable.

In the present survey no patient was considered satisfactory in the presence of thirst or pruritus. Polyuria up to three litres of urine in the twenty-four hours was condoned as it did not obtrude itself on the patient's consciousness nor manifest itself as nocturia. The quantity of urine passed in the twenty-four hours was very similar for free and control groups.

Glycosuria must not be sufficient, relative to the carbohydrate intake, to lead to an inadequate carbohydrate utilisation. It has been recognised for a long time that carbohydrate utilisation can occur in the presence of hyperglycaemia, and, indeed, Soskin and Levine (1946) and Himsworth (1949) believed that hyperglycaemia acted as an aid to utilisation. Adequate carbohydrate utilisation ensures freedom from ketosis, satisfactory physical energy, an adequate weight, and normal growth and development in children.

These criteria for control have been met in the treatment of the free diet group, despite glycosuria.

If free diets lead to a carbohydrate utilisation in excess of the body requirement, obesity may result.

In this survey, only those with a history of precursory obesity tended to become overweight under such treatment.

The question of the relationship between hyperglycaemia and repeated pyogenic infections deserves separate consideration. Formerly it was thought that increased blood and tissue sugar contents favoured the growth of bacteria, particularly staphylococci. However, more recent experiments on bacterial cultures in vitro do not support this view. Recent ideas relate the lowered resistance to infection in diabetes to malnutrition and a lowered liver glycogen content and ketosis, associated with inadequate carbohydrate utilisation. Rudy and Hoffman (1942) suggest a contributory deficiency of the vitamin B complex. Tolstoi (1943) noted good healing of surgical wounds despite continuous post-operative hyperglycaemia and found no increased liability to pyogenic infections in his glycosuric patients, where ample carbohydrate utilisation was assured by the administration of adequate insulin.

There has not been a remarkably high incidence of septic cuts, boils or styes in the free diet group during the present survey. However, four injection abscesses have occurred. The reason for this increased incidence of injection abscess, in the absence of liability to other forms of sepsis, is obscure, unless it be due to two insulin injections daily which double the risk of the introduction of infection.

Hyperglycaemia is associated with wide fluctuations of blood sugar. Theoretically, therefore, one might expect frequent insulin reactions among patients on free diets. This, however, is not the case where insulin is used wisely. Reactions are indeed more common when an attempt to follow a régime of normoglycaemia is made, and little margin is allowed for exercise or excitement. Free diets allow the flexibility of circumstance, more food being taken during days of hard exercise. The harmful effects of hypoglycaemia, particularly on the central nervous system and cardiovascular system, are well known, and any successful régime of diabetic treatment must minimise the risk of reactions as far as possible.

2. HYPERGLYCAEMIA AND THE PANCREAS.

Insulin, which has such a beneficial effect in the treatment of diabetes mellitus, is the internal secretion of the pancreas. At first, a simple deficiency of insulin was postulated as the cause of diabetes, just as a deficiency of thyroid leads to myxoedema, and it was hoped that insulin would restore patients to virtual normality.

However, it is impossible to provide physiological replacement therapy in diabetes, as subcutaneous injections of insulin are absorbed into the systemic circulation, whereas the normal route is via the portal circulation. Also, the sensitively determined variability of the insulin secretion of a normal pancreas, resulting in a balanced carbohydrate metabolism, with normal blood sugar levels, cannot be achieved by insulin injections. Furthermore, insulin acts in an environment influenced by the endocrine system as a whole, which determines the sensitivity of the tissues to insulin and evidence exists that the endocrine background may be abnormal in diabetes. The treatment of diabetes with insulin has served only to unmask, because of the longer lives of the patients, the high incidence of degenerative complications associated with the disease.

The production of diabetes by pancreatectomy in the laboratory of Von Mering and Minkowski (1889), and the discovery of the pancreatic extract, insulin, by Banting and Best (1922) with its dramatic therapeutic value, placed the pancreas in the forefront of diabetic pathology. More recent work also emphasises the importance of the pancreas, namely the discovery of alloxan diabetes by Shaw-Dunn and McLetchie (1943), in which a highly selective destruction of the beta cells of the islets of Langerhans is followed by full-blown diabetes, and the production of diabetes by total pancreatectomy, in man, reported by Ricketts (1947).

Allen (1922) demonstrated that the pathological change in the pancreas, which accompanied downward progress and which ultimately led to complete disappearance of the beta cells of the islets in experimental animals, was hydropic degeneration. This could be induced and halted at will by suitable feeding, and thus could be demonstrated positively as an anatomic breakdown of islet cells due to functional overstrain.

The occurrence of post-prandial hyperglycaemia and a rebound hypoglycaemia in certain patients following partial gastrectomy was described by Gilbert and Dunlop (1947). One of the patients exhibiting

this syndrome subsequently died and, at post mortem, the islet cells showed hypertrophy. This finding was thought to be a result of stimulation of the pancreas by hyperglycaemia.

Haist, Campbell and Best (1940) found that pituitary diabetes could be prevented by large doses of insulin given simultaneously, or by low calorie or high fat diets.

Continuing studies in this field, Lukens, Dohan and Wolcott (1943) found that the treatment of pituitary diabetes in the cat with phlorhizin, if begun soon enough, led to recovery of the animals. These writers pointed out that the only clear similarity of the action of insulin, low diets and phlorhizin was in abolishing hyperglycaemia. They suggested, therefore, that the level of the blood sugar might play an important part in the pathogenesis of diabetes. Finally, Dohan and Lukens (1947) have shown that, if a pronounced hyperglycaemia is maintained in normal cats by repeated intraperitoneal injections of glucose, degeneration of the islet cells and permanent diabetes may follow. They concluded, "these findings support the hypothesis that a sustained elevation of blood glucose may, under certain conditions, lead to the production of damage to the islands of Langerhans in this species".

Himsworth (1949) suggested that there is strong evidence that hyperglycaemia per se can produce a secondary insulin deficiency by pancreatic damage. However, he also quoted Peters, "no condition approaching the gravity of the disorders of metabolism encountered in severe spontaneous human diabetes has been produced in man by destruction or removal of the pancreas".

Warren (1930) found a perfectly normal pancreas in 27% of diabetic patients while many of the remaining 73% showed lesions of insufficient extent to account for the diabetes.

On the other hand there is strong evidence to suggest that the anterior pituitary and suprarenal glands play an important part in diabetes mellitus.

Houssay (1936) found that the removal of the pituitary produced a hypersensitivity to insulin which could be removed by the injection of anterior pituitary extracts. Depancreatised dogs lived longer if the pituitary was also removed, and a return of severe diabetes was produced by injections of anterior pituitary extracts. He obtained a phase of insulin resistance and glycosuria with spontaneous recovery by injecting anterior pituitary extracts into intact animals. Young (1937) proved that permanent diabetes could result from continuous anterior pituitary injections into adult dogs.

Similar work has been carried out on the suprarenals.

Long and Lukens (1934) found that adrenalectomy ameliorated pancreatic diabetes in the cat just as did hypophysectomy in the dog. The converse of these experiments, the induction of diabetes by the injection of adrenal cortical extracts, was not accomplished in normal animals, but accentuation of the diabetes of partially depancreatised rats by the administration of certain fractions of adrenal cortical extract was achieved by Long and his co-workers (1940) and by Ingle (1940). I have heard of a patient with diabetes mellitus requiring 50 units of insulin daily who developed Addison's disease and thereafter was controlled on only six units.

The principle of pancreatic rest was applied to human diabetes by Allen who proved that undernutrition, by strict dieting, led to an improvement in carbohydrate tolerance, which, however, was lost if, by reason of dietary excess, hyperglycaemia were allowed. Undernutrition and sugar-free urine were corner-stones in the pre-insulin treatment of diabetes.

However, Allen (1922) in reporting his results of using insulin in the treatment of diabetics stated - "The experience thus far gives the distinct impression

that if decline of tolerance results from glycosuria and hyperglycaemia under insulin treatment, it is at least decidedly less marked and rapid than under similar conditions without insulin".

Brush (1944) described a method of treatment for acute early cases of diabetes in children, using weighed diets and large doses of insulin, so that for the first three to four weeks the blood sugars were kept low, just above the reaction level. He found that improvement occurred and that, after about four weeks, the children could be discharged home on 2-8 units of insulin only, in the day. The régime, however, did not modify long-term results and the children came to require the usual high doses of insulin. The phenomenon is frequently noted in hospital when, following diabetic coma and large doses of insulin, the diabetic state becomes temporarily less severe. Brush suggested that the remission was due to functional recovery of the pancreas when it was freed from the stimulus of hyperglycaemia. However, another possible explanation lies in anterior pituitary inhibition from excess of insulin.

Lukens (1947) advocated prompt treatment and the maintenance of normal sugars in early acute cases of diabetes, especially in children. "Sparing the pancreas" in such early cases has been widely advised

and other exponents include such varied writers as Payne (1947), Mosenthal (1948) and Micks (1943). Once diabetes is well established - and Lawrence (1944) indicates that despite dietetic treatment, most diabetics become worse till a stage of maximal diabetes is reached - the importance of sparing the pancreas is less evident. In such patients, Mosenthal (1948) allowed mild hyperglycaemia.

The Editorial of the American Journal of Digestive Diseases (1949) raised no objection to the régime of Tolstoi, where the daily insulin requirement was above forty units. According to Ricketts (1947) forty units a day was the maximal requirement of insulin found in diabetes in man following pancreatectomy. However, the hypothesis that, in patients requiring more than forty units of insulin daily, the pancreas is no longer functioning, is open to several objections. A pancreatectomised "normal" adult has lost the external secretion of the pancreas as well, and inference even from human to human is not therefore sound. Also, the argument does not take into account the important element of insulin inhibition.

The truth of the matter is that, in human living diabetics, there is no means of telling to what extent diabetes is due to insulin lack and to what

extent to insulin inhibition. Low calorie diets, it is argued, "spare the pancreas" but, at the same time, the effect might equally well be due to anterior pituitary inhibition.

3. HYPERGLYCAEMIA AND DEGENERATIVE COMPLICATIONS.

Are hyperglycaemia and degenerative complications different expressions of a single constitutional defect, or does hyperglycaemia cause or facilitate the development of degenerative complications? The degenerative complications associated with diabetes are retinitis, peripheral vascular disease, Kimmelstiel-Wilson kidney, and other expressions of vascular disease such as angina. Diabetic cataract and diabetic neuropathy complete the group of degenerative lesions.

These disabilities usually appear after years of manifest diabetes. However, occasional patients come complaining of failure of vision or intermittent claudication in the absence of hyperglycaemia and glycosuria or clinical symptoms of diabetes. These patients have, however, a diabetic blood sugar curve and frequently develop frank diabetes later. The constant presence of hyperglycaemia as a requirement for the development of retinitis or peripheral vascular disease is thus disproved. In addition, many years of hyperglycaemia do not invariably lead to degenerative complications and, on the other hand, apparently excellent diabetic control by no means guarantees the avoidance of degenerative disease.

One cannot promise freedom from complications in return for the faithful adherence to a strict dietetic régime. Finally, the question arises whether such a method diminishes the risk of complications or not.

The evidence must be based on long-term results and a short survey of the literature will be made under the headings - Retinitis, Peripheral Vascular Disease, Kimmelstiel-Wilson kidney, Diabetic Neuropathy, Cataract. No contribution to this aspect of the problem can be made by the present survey.

RETINITIS.

A tentative suggestion was made by Elwyn (1941) that a persistently increased sugar level in the blood influenced the terminal vessel units in some unknown manner, resulting in a chronic state of prestasis, with repeated haemorrhages into the retina.

No obvious correlation with blood sugar levels or insulin dosage was noted by Waite and Beetham (1935) who found that deep retinal haemorrhages bore a relation to the duration of diabetes.

Leopold (1942) found diabetic retinitis as frequently in well controlled as in poorly controlled patients.

The most meticulous diabetic care seldom resulted

in any semblance of a reversal of the pathologic process in the eye or even of a staying of the process in the experience of Anderson (1942).

Gifford (1943), the guest speaker at a Conference on diabetic retinitis, stated that it had not been his experience that the usual control of diabetes by insulin or diet had much effect on the course of retinopathy.

In a survey of twenty-five years' experience of diabetic retinitis, Wagener (1945) stressed the dominant rôle played by the duration of the diabetes but was not convinced that good diabetic control had either a preventive or regressive value in diabetic retinitis. "It becomes increasingly apparent that something inherent in the diabetes is responsible for the development of retinopathy. Yet no specific factor has been proved to be accountable and hence preventable or removable."

The progress of diabetic retinitis was described by Ballantyne (1947) as follows. First of all, micro-aneurysms appear on the retinal capillaries, with or without punctate haemorrhages and punctate exudates. The haemorrhages and exudates increase in number and size. Finally, the veins are involved with haemorrhage into the retina and vitreous, massive exudates and detachment of the retina. The

thread which runs throughout and links up one stage with another is an affection of the retinal capillaries and veins.

He concluded, "The outstanding paradox is the lack of any apparent relation between the severity of the diabetes, the control of the condition by treatment, and the presence or absence of retinal changes. Again and again in these investigations one finds the case clinically described as mild and well controlled, and sugar-free from the first discovery of the disorder, while notable and progressive retinal changes are present; and the converse, severe diabetes badly controlled but with normal fundi, is also a frequent occurrence. .... There seems only to be general consent to the view that the duration of the diabetes is of the greatest consequence, and retinitis is commonest between the ages of fifty and seventy years."

This view has been confirmed in the Dietetic Out-patient Department of the Royal Infirmary. Croom and Scott (1949) reporting on sixty diabetics stated, "there did not appear to be any correlation between the retinal changes and the severity or control of the diabetic state".

PERIPHERAL VASCULAR DISEASE.

The incidence of degenerative complications in diabetes is high. Two articles from the literature on their occurrence, even in young people, will first be considered.

Priscilla White (1947) reported upon 249 patients who had become diabetic in childhood and had survived twenty or more years of diabetes. She gave the following figures for the incidence of degenerative complications in those examined -

Retinal haemorrhage	65%
Retinal exudate	50%
Retinitis proliferans	8%
Arteriosclerosis	70%
Albuminuria	35%
Cataract	1.6%
Neuritis	15%

A follow-up of 123 patients who had become diabetic in childhood before 1932 was given by Chute (1948). He found that all of the twenty-four patients who had survived twenty years of diabetes showed pronounced degenerative disease, and that complications were present to a lesser degree in a group surviving fifteen years.

Many theories of causation have been put forward. Rabinowitch (1935 and 1944) believed high carbohydrate-low fat diets reduced the incidence of degenerative complications by lowering the blood cholesterol levels.

Mosenthal (1943) advocated the conservation of body protein as the most important factor.

However, the present discussion is concerned with the possible harmful effects of hyperglycaemia in this connection. Joslin (1947) thought that the carefully controlled young patient was the one in best condition after twenty years. Boyd, Jackson and Allen (1942) reported their findings in sixty-nine young adult patients previously under their care as children and later recalled for examination. Sixty of these were of at least five years' duration and forty-two had been diabetic for ten years or more. The degree of control of the disease was termed poor if hyperglycaemia was fairly constant. Among those who were poorly controlled were twelve dwarfs, eleven cases of retarded maturation, six cases of retinal haemorrhage and six cases of cataract. Such lesions were not present in patients whose adherence to the prescribed régime had been adequate. The complications mentioned including dwarfism and delayed puberty in poorly controlled patients suggest that the hyperglycaemia was accompanied by inadequate utilisation of carbohydrate.

In 1947, Boyd expressed the view that a physician "can expect confidently that his young diabetic patient will make good progress with freedom from

degenerative lesions so long as his diabetic state is kept approximately under physiologic levels of control".

In a survey of diabetics observed for ten years or longer, Naide (1945) studied the occurrence of arteriosclerosis of the feet. He found that 46% of 35 patients who were poorly controlled had peripheral arteriosclerosis. This was compared to 35% with sclerosis in the 54 well-controlled patients, a small difference.

Dolger (1947) in a review of 200 patients followed up for twenty-five years found a high incidence of degenerative complications; not one escaped retinal haemorrhage, regardless of the age of onset, severity of the diabetes, or the type of treatment used. The level of diabetic control made no appreciable difference to the development of complications but a definite increase was noted with the duration of diabetes and the elderly developed complications in a shorter time interval than the younger patients. Dolger believed that the obvious disturbance of carbohydrate metabolism in diabetes had overshadowed the little understood, generalised nature of the disease and he found that complications in glycosuric patients arose neither sooner nor more frequently than in the aglycosuric.

In an investigation of possible etiological factors in diabetic vascular disease, Herzstein and Weinroth (1945) found also that age and, more particularly, the duration of the diabetes were the only clear-cut influences. Their figures for vascular disease in relation to hyperglycaemia are interesting. Patients were standardised as follows:-

1. Good control - Freedom from glycosuria.
2. Fair control - Intermittent glycosuria.
3. Poor control - Persistent glycosuria and hyperglycaemia.

The results were given in a table, reproduced below.

Control of Diabetes and Peripheral Vascular Disease.

	Control	Number of Patients	Peripheral Vascular Disease present	% with Peripheral Vascular Disease
1	Good	155	78	50%
2	Fair	48	29	60%
3	Poor	42	14	33%

The lower incidence in poorly controlled diabetics was not due to their being predominantly young.

The longest follow-up of the free diet method was reported by Lichtenstein (1945). After ten years, in 169 cases of diabetes in children and young persons up to the age of twenty-nine, he found no increased incidence of degenerative complications.

KIMMELSTIEL-WILSON KIDNEY.

A pure lesion of intercapillary glomerulosclerosis in cases of diabetes mellitus was described by Kimmelstiel and Wilson (1936). Clinically the lesion may be associated with the nephrotic type of oedema, gross albuminuria, hypertension, renal decompensation and retinitis.

Laipply, Eitzen and Dutra (1944) stated that, in their view, intercapillary glomerulosclerosis was a more specific finding than hyalinisation of the islets of Langerhans in diabetics at post mortem.

Goodof (1945) found that the kidney lesions occurred more often in older age groups and more frequently with increased duration of the diabetes. He gave no figures for the correlation between hyperglycaemia and kidney lesions.

CATARACT.

True diabetic cataract occurring in young people is very rare. Diabetics tend to develop a senile type of cataract earlier than normal persons. Although, when patients are first stabilised on insulin, vision is sometimes temporarily impaired, presumably by the effect of changes in the blood sugar level on the lens, there is no proof that

fluctuating blood sugar levels predispose to the development of lens opacities.

Boyd, Jackson and Allen (1942), in the 69 young adult patients already mentioned, found six cases of cataract and twenty further cases of minor subcapsular opacities among those with poorly controlled diabetes. In contrast to their findings, Karlström (1941) reported 38 children treated with free diets to be free of cataract.

DIABETIC NEUROPATHY.

Rundles (1945) reported his observations on 125 cases of diabetic neuropathy. He believed that most diabetics who developed organic disease of the peripheral nerves to have had antecedent periods, usually of months or years, of grossly neglected or poorly managed diabetic treatment. He amplified his definition of gross neglect as resulting in clinical symptoms of thirst and polyuria and loss of weight.

There is thus evidence to suggest that poor clinical control of diabetes with inadequate carbohydrate utilisation may contribute towards the production of diabetic neuritis. It is not clear, however, that hyperglycaemia alone is to blame.

Summary.

The experience gained by this investigation supports the claims made by writers on liberal diets that satisfactory clinical results may be obtained despite the presence of hyperglycaemia and glycosuria.

The place of the pancreas in the pathology of Diabetes Mellitus and the possible harmful effect of hyperglycaemia upon its function are not absolutely clear. Ricketts (1947) gives a summary of the present conception. "The pancreas is in all likelihood involved to some degree, though perhaps not exclusively, in at least a large number of patients with diabetes mellitus. ..... The evidence in animals indicates that both functional and structural injury to the beta cells may result if the blood sugar is allowed to remain high, certainly during the incipient stages of diabetes and possibly even after the disease has become fixed. The evidence in man, while not so definitive, points in the same direction."

The consensus of opinion is that hyperglycaemia plays no part in the production of retinitis. Literature on its relation to cataract or kidney disease is inconclusive. As regards peripheral vascular disease and, more particularly, neuritis,

there is evidence to suggest that poor clinical control plays a part. There is, however, no clear-cut support of the theory that hyperglycaemia is harmful per se in the presence of good clinical control and adequate carbohydrate utilisation.

Chapter X.

SUMMARY AND CONCLUSIONS.

Undernutrition and normoglycaemia were fundamental concepts in the treatment of diabetes mellitus in the pre-insulin era. Strict dietetic régimes produced a substantial amelioration of the disease in many patients and Allen postulated that loss of weight with an associated diminution in total metabolism was the basis for this phenomenon.

That subcaloric diets result in tiredness, retarded growth and sexual development and poor nutrition with diminished resistance to infections is well recognised at the present time and inhibition of the anterior pituitary gland is thought to be the underlying mechanism. The depression of the diabetogenic effect of the anterior pituitary cannot be achieved without concomitant inhibition of growth and sexual functions.

The discovery of insulin introduced the possibility of nourishing diabetics. However, the principle of undernutrition continued to hold pride of place in the treatment of diabetes because of the difficulty in attaining full nutrition without incurring the risk of hyperglycaemia. Insulin was

employed merely as an adjunct to dietetic measures and in very moderate doses.

However, there has been a slow and consistent tendency over many years to prescribe larger diets with ever increasing quantities of carbohydrate. Very varied methods are in use at present. Sub-caloric diets involving the weighing of carbohydrate, fat and protein are still widely advocated. Other clinics allow free choice of foods consisting of protein and fat and advise weighing only of carbohydrates. The portions of carbohydrate may be estimated by the use of models in centres where less accurate diets are permitted. The present investigation is concerned with free diets except for the avoidance of concentrated carbohydrates in the form of sugar, jam and sweets.

Among the advantages claimed for liberal diets are greater energy, freedom from hunger, fewer insulin reactions and improved nutrition and resistance to infections. These simpler methods are preferable from the psychological point of view. Patients are able to live practically normal lives. They are quick to grasp the fundamental principles of treatment, including regularity of meals and insulin injections, the prompt treatment of incipient reactions and the early recognition of thirst, polyuria or a

positive acetone test as indicative of poor control. They are not required to test for urine sugar and do not become worried over a complicated system of dietary exchanges.

Such methods undoubtedly reduce the rate of defaulting from the diabetic clinic. This, in itself, is a great advantage as regular supervision is essential for the successful treatment of diabetics. Finally, there are many rebellious spirits who adopt a free régime despite careful dietary instructions. Quite a number of those included in the present survey were considered unsatisfactory in the Dietetic Clinic for this reason. On a free régime with insulin administration adapted accordingly they have kept very well.

The short-term results of a liberal form of diabetic treatment have been presented, the maximum duration of treatment being three and a half years. Concentrated carbohydrates in the form of sugar, jam and sweets were avoided but otherwise patients were permitted free choice of foodstuffs. Insulin was prescribed to maintain clinical control. The requirements were slightly in excess of those for dietetically treated patients and the total dose was divided into two injections daily in order to minimise the risk of reactions. No attempt was made to

obtain freedom from glycosuria provided clinical results were satisfactory.

The control group, studied for comparison, was selected from co-operative members of the Dietetic Clinic where subcaloric diets are used with the weighing of carbohydrate, protein and fat. The diets contain relatively more fat than normal diets. The method employed aims at a reduction of glycosuria to the minimum compatible with freedom from insulin reactions.

Apart from those patients classed as failures on a liberal régime and given controlled diets, freedom from thirst, pruritus and acetonuria was achieved. An asymptomatic degree of polyuria was permitted and it was interesting to find that the urine outputs of free diet and control patients were much the same.

Patients on free diets were well nourished and, on the whole, were physically stronger than those on restricted diets. Obesity occurred only in those with a marked history of overweight prior to the onset of diabetes. Growth and development were very satisfactory and there was a high incidence of pregnancy in young married women. Pyogenic complications were not frequent but four injection abscesses occurred in patients having two injections of insulin daily, a method which carries with it a double risk

of infection. Diabetic coma developed in two patients. Four exhibited diabetic retinitis and one an early senile cataract.

Among patients of the control group one sensed a greater concern over their disability. A few complained of hunger and a considerable proportion were below their average weight of the past and were more easily tired than before the onset of diabetes.

Glycosuria was greater in the free diet group, yet the twenty-four hour carbohydrate utilisation was superior to that of the controls. Hyperglycaemia was more frequent and severe in patients receiving liberal diets.

Five young people of puberty age and two adults became uncontrollable on free diets. Two patients were given regulated intakes because of frequent insulin reactions. Two women with marked histories of precursory obesity were changed to restricted diets because of a renewed tendency in this direction. The remaining forty-four patients are being treated on free diets at present but it is possible that in the future increasing insulin requirements may necessitate a change to controlled diets in some of these.

To obtain full growth and normal sexual development in young diabetics a relatively high calorie intake and large doses of insulin are required.

Diabetes Mellitus is known to increase in severity at puberty and the disease became too severe in those children of this age group studied to permit the continued use of free diets. A change to regulated diets slightly less than those previously chosen was therefore made.

In the future selection of patients for liberal diets, it would be wise to omit young people of puberty age, and to extend the exclusion of obese adults to those, even if underweight when first seen, with a marked history of obesity prior to the onset of diabetes. The present survey did not include a sufficient number of young children for any statement to be made regarding the employment of free diets for them. Several articles have praised the method for children. The majority of adults with no history of precursory obesity have kept very well on free diets and many feel as strong and healthy as they ever did in the past.

The virtually free diet outlined cuts across the old-established principles of undernutrition and normoglycaemia. The more liberal the régime the happier the patient but the greater the liability to hyperglycaemia. It must be emphasised, however, that the achievement of consistent normoglycaemia is very difficult and it demands an even stricter régime than that employed in the Dietetic Clinic.

In this investigation, hyperglycaemia has proved compatible with good clinical control in the broadest sense.

Restricted diets undoubtedly modify the severity of diabetes but there is evidence to suggest that this is achieved at the expense of anterior pituitary inhibition and the truth of the argument that it is entirely due to a "sparing of the pancreas" by the avoidance of hyperglycaemia is questionable. The practical issue is whether a régime which condones hyperglycaemia leads to deterioration of sufficient degree to outweigh the advantages of the liberal method. Allen's principle cannot be disregarded in severe diabetes but, nevertheless, the majority of non-obese adults have proved readily controllable on free diets to date. Ever increasing insulin requirements may make dietary restriction necessary in the future. The duration of the survey is not long enough to give a final answer to the problem or to that of the possible increased incidence of degenerative complications due to hyperglycaemia.

Examination of the literature reveals a confusion between hyperglycaemia and inadequate clinical control as manifest in thirst, polyuria and ketosis. The two have been considered synonymous which, undoubtedly, is not the case. In the presence of

adequate carbohydrate utilisation and full control of diabetic symptoms, there is no convincing evidence that hyperglycaemia is harmful. The age of the patients and the duration of their diabetes are the only two clear-cut etiological factors in the production of degenerative disease. Arguments for the harmfulness of hyperglycaemia per se are thus inferential and inconclusive.

Liberal diets may prove of value in the treatment of adult diabetics requiring insulin and who give no history of precursory obesity. Over the period covered by this survey, clinical control in its broadest sense has been achieved and maintained in practically every patient of this category. Therefore, provided no marked deterioration occurs in the future and the incidence of degenerative complications is not increased, the evidence would support a greater liberality of outlook in the treatment of this form of diabetes mellitus.

ACKNOWLEDGMENTS.

I wish to thank Professor D. M. Dunlop and the members of his staff for their advice and encouragement throughout my work.

The survey was carried out during the tenure of a Crichton Research Scholarship and a Carnegie Senior Scholarship, held consecutively; I am indebted to the trustees of these sources for their financial support.

REFERENCES.

Adlersberg and Porges (1926): Quoted from The Treatment of Diabetes Mellitus, by Joslin, E. P. and others, London, 1947, eighth edition, page 352.

Allen, F. M. (1914): J. Amer. Med. Ass., 63, 939.

Allen, F. M. (1917): Amer. J. Med. Sci., 153, 313.

Allen, F. M., and Sherrill, J. W. (1922): J. Metab. Res., 2, 803.

Allen, F. M. (1923): J. Metab. Res., 3, 61.

Anderson, G. E. (1942): Arch. Ophthal., 28, 679.

Ballantyne, A. J. (1947): Trans. Ophthal. Soc. U.K., 66, 503.

Banting, F. G., and Best, C. H. (1922): J. Lab. Clin. Med., 7, 464.

Barach, J. H. (1928): Ann. Med. Hist., 10, 387.

Boyd, J. D., and Nelson, M. V. (1928): Amer. J. Dis. Child., 35, 753.

Boyd, J. D., and Kantrow, A. H. (1938): Amer. J. Dis. Child., 55, 460.

Boyd, J. D., Jackson, R. L., and Allen, J. H. (1942): J. Amer. Med. Ass., 118, 694.

Boyd, J. D. (1947): Med. Clin. N. Amer., 31, 279.

Brown, G. D., and Thompson, W. H. (1940): Amer. J. Dis. Child., 59, 238.

Brush, J. M. (1944): Amer. J. Dis. Child., 67, 429.

Chute, A. L. (1948): Amer. J. Dis. Child., 75, 1.

Colwell, A. R. (1947): Med. Clin. N. Amer., 31, 327.

Croom, J. H., and Scott, G. I. (1949): Lancet, 1, 555.

A Doctor (1946): Practitioner, 157, 440.

A Doctor (1949): Lancet, 1, 116.

Dohan, F. C., and Lukens, F. D. W. (1947): Science, 105, 183.

Dolger, H. (1947): J. Amer. Med. Ass., 134, 1289.

Editorial: Amer. J. Digest. Dis. (1949), 16, 30.

Ellis, R. W. B. (1945): Arch. Dis. Childh., 20, 97.

Elwyn, H. (1941): Arch. Ophthal., 25, 139.

Fischer, A. E., Mackler, H. S., and Marks, H. H. (1942): Amer. J. Dis. Child., 64, 413.

Fischer, A. E., and Dolger, H. (1946): Arch. Intern. Med., 78, 711.

Geyelin, H. R. (1926): Atlant. Med. J., 29, 825.

Geyelin, H. R. (1935): J. Amer. Med. Ass., 104, 1203.

Gifford, S. R. (1943): Amer. J. Digest. Dis., 10, 329.

Gilbert, J. A. L., and Dunlop, D. M. (1947): Brit. Med. J., 2, 330.

Goodof, I. I. (1945): Ann. Intern. Med., 22, 373.

Gray, P. A., and Sansum, W. D. (1933): J. Amer. Med. Ass., 100, 1580.

Greenblatt, R. B., and Nieburgs, H. E. (1947): Med. Clin. N. Amer., 31, 712.

Guthrie, D. (1945): A History of Medicine, Thomas Nelson & Sons, Ltd., Edinburgh.

Haist, R. E., Campbell, J., and Best, C. H. (1940): New Engl. J. Med., 223, 607.

Harris, H. A. (1947): Child Health and Development, edited by R. W. B. Ellis, London, page 148.

Hagedorn, H. C., Jensen, B. N., Krarup, N. B., and Wodstrup, K. (1936): J. Amer. Med. Ass., 106, 177.

Herzstein, J., and Weinroth, L. A. (1945): Arch. Intern. Med., 76, 34.

Himsworth, H. P. (1936): Lancet, 1, 127.

Himsworth, H. P. (1939): Lancet, 2, 171.

Himsworth, H. P. (1940): Brit. Med. J., 1, 719.

Himsworth, H. P. (1949): Lancet, 1, 465.

Houssay, B. A. (1936): New Engl. J. Med., 214, 971.

Ingle, D. J. (1940): Proc. Soc. Exp. Biol. & Med., 44, 176.

Jackson, R. L., and McIntosh, C. B. (1945): Amer. J. Dis. Child., 70, 307.

Jackson, R. L., and Kelly, H. G. (1946): J. Pediat., 29, 316.

Jacobs, E. C. (1948): J. Clin. Endocrinol., 8, 227.

John, H. J. (1935): Amer. J. Digest. Dis., 1, 855.

Joslin, E. P., Root, H. F., and White, P. (1925): J. Amer. Med. Ass., 85, 420.

Joslin, E. P. (1947):
(a) Advocating strict diabetic control. Med. Clin. N. Amer., 31, 259.
(b) Regarding insulin sensitivity tests. The Treatment of Diabetes Mellitus, edited by Joslin, E. P., and others, London, 1947, eighth edition, page 143.
(c) Regarding the ideal diabetic diet. The Treatment of Diabetes Mellitus, edited by Joslin, E. P., and others, London, 1947, eighth edition, page 259.

Karlström (1941): Quoted from The Treatment of Diabetes Mellitus, edited by Joslin, E. P., and others, London, 1947, eighth edition, page 761.

Kimmelstiel, P., and Wilson, C. (1936): Amer. J. Path., 12, 83.

Klatskin, G. (1938): J. Clin. Invest., 17, 745.

Laipply, T. C., Eitzen, O., and Dutra, F. R. (1944): Arch. Intern. Med., 74, 354.

Landau, R. L. et al. (1948): J. Clin. Endocrinol., 8, 133.

Lawrence, R. D. (1944): The Diabetic Life, London, 1944, thirteenth edition, page 86.

Leopold, I. H. (1942): Arch. Ophthal., 27, 422.

Lichtenstein, A. (1938): J. Pediat., 12, 183.

Lichtenstein, A. (1945): Acta Pediat., 32, 556.

Long, C. N. H., and Lukens, F. D. W. (1934): Science, 79, 569.

Long, C. N. H., Katzin, B., and Fry, E. G. (1940): Endocrinology, 26, 309.

Loughlin, W. C., and Mosenthal, H. O. (1944): Amer. J. Dis. Child., 68, 13.

Lukens, F. D. W., Dohan, F. C., and Wolcott, M. W. (1943): Endocrinology, 32, 475.

Lukens, F. D. W. (1947): Med. Clin. N. Amer., 31, 387.

MacBryde, C. H. (1936): J. Clin. Invest., 15, 577.

McGavin, A. P., Schultz, E., Peden, G. W., and Bowen, B. D. (1940): New Engl. J. Med., 223, 119.

von Mering and Minkowski (1889): Quoted from The Treatment of Diabetes Mellitus, edited by Joslin, E. P., and others, London, 1947, eighth edition, page 86.

Micks, R. H. (1943): Brit. Med. J., 1, 598.

Micks, R. H. (1944): Brit. Med. J., 2, 784.

Mirsky, I. A. (1942): J. Amer. Med. Ass., 118, 690.

Mosenthal, H. O. (1943): Symposium on Diabetic Retinitis, Amer. J. Digest. Dis., 10, 329.

Mosenthal, H. O. (1948): Ann. Intern. Med., 29, 79.

Naide, M. (1945): Amer. J. Med. Sci., 209, 23.

Newburgh, L. H., and Marsh, P. L. (1920): Arch. Intern. Med., 26, 647.

Payne, W. W. (1938): Lancet, 2, 195.

Payne, W. W. (1947): Diseases of Children, edited by Paterson, D., and Moncrieff, A., London, fourth edition, Volume I, page 378.

Peters, J. P., and Van Slyke, D. D. (1931): Quoted by Tolstoi (1943).

Petren (1922): Quoted from Clinical Diabetes Mellitus and Hyperinsulinism, by Wilder, R. M., Philadelphia and London, page 109.

Rabinowitch, I. M., and Bazin, E. V. (1929): Arch. Dis. Childh., 4, 125.

Rabinowitch, I. M. (1930): Canad. Med. Ass. J., 23, 489.

Rabinowitch, I. M. (1935): Ann. Intern. Med., 8, 1436.

Rabinowitch, I. M. (1944): J. Canad. Med. Ass., 51, 300.

Reiner, L., Searle, D. S., and Lang, E. H. (1939): Proc. Soc. Exp. Biol. & Med., 40, 171.

Ricketts, H. T. (1947): Med. Clin. N. Amer., 31, 267.

Root, H. F., and Marble, A. (1947): The Treatment of Diabetes Mellitus, edited by Joslin, E. P., and others, London, 1947, eighth edition, page 420.

Rudy, A., and Hoffman, R. (1942): New Engl. J. Med., 227, 893.

Rundles, R. W. (1945): Medicine, 24, 111.

Sansum, W. D., Blatherwick, N. R., and Bowden, R. (1926): J. Amer. Med. Ass., 86, 178.

Shaw-Dunn, J. S., and McLetchie, N. G. B. (1943): Lancet, 2, 384.

Soskin, S., and Levine, R. (1938): J. Amer. Med. Ass., 110, 768.

Soskin, S., and Levine, R. (1946): Carbohydrate Metabolism, Chicago.
(a) Gluconeogenesis from fat, page 139.
(b) Hyperglycaemia and carbohydrate utilisation, pages 185 and 253.

Spencer, H. (1928): Amer. J. Dis. Child., 36, 502.

Sprague, R. G. (1947): Med. Clin. N. Amer., 31, 445.

Stolte, K. (1931): Med. Klin., 27, 831.

Talbot, N. B., and Sobel, E. H. (1947): Advances in Pediatrics, Volume 2, page 238.

Tolstoi, E., and Weber, F. C. (1939): Arch. Intern. Med., 64, 91.

Tolstoi, E., and Weber, F. C. (1940): Arch. Intern. Med., 66, 670.

Tolstoi, E., Almy, T. P., and Toscani, V. (1942): Ann. Intern. Med., 16, 893.

Tolstoi, E. (1943): Amer. J. Digest. Dis., 10, 247.

Wagener, H. P. (1945): Collected Papers of the Mayo Clinic and the Mayo Foundation, 37, 414.

Waite, J. H., and Beetham, W. P. (1935): New Engl. J. Med., 212, 429.

Warren, Shields (1930): Quoted by Ricketts, 1947.

De Wesselow, O. L. V., and Griffiths, W. J. (1938): Quart. J. Med., 7, 17.

White, P. (1947): The Treatment of Diabetes Mellitus edited by Joslin, E. P., and others, London, 1947, eighth edition, page 741.

Wilder, R. M. (1941): Clinical Diabetes Mellitus and Hyperinsulinism, Philadelphia and London, page 124.

Young, B. A. (1949): Lancet, 1, 125.

Young, F. G. (1937): Lancet, 2, 371.

A P P E N D I X.

APPENDIX.

Part I.

Estimation of free diets was difficult because none of the patients possessed scales. However, the patients whose diets were included in the survey were very co-operative and wrote out careful accounts of all the food taken and milk or beer consumed, over the period of a week. Quantities of the foods eaten were stated. Conversion figures were based upon those given in The Chemical Composition of Foods, by R. A. McCance and E. M. Widdowson, London, 1946, second edition. The estimations of the carbohydrate, protein and fat contents of the diets will be considered separately. Exactly the same methods were used for each patient, including the assessment of the diets actually taken by those of the control group.

CONVERSION TABLE FOR CARBOHYDRATE FOODS.

	Item	Quantity	Approximate Carbohydrate Content. Grammes
1	Bread	1 medium slice of brown bread	15
		1 medium slice of National loaf	20
		1 penny roll	30
2	Potato	1 boiled potato, the size of a hen's egg, or	
		1 tablespoonful of boiled potato	15
3	Porridge	1 tablespoonful of cooked porridge	5
4	Cornflakes	1 teacupful of cornflakes	20
5	Oatcake	1 medium sized oatcake	7
6	Biscuit	1 medium sized biscuit	7
7	Ryvita	1 ryvita	7
8	Meat Pie	1 fourpenny pie	30
9	Scone	1 penny scone or bun	20
10	Gingerbread	1 slice of gingerbread	15
11	Cake	1 cake or tart	25
12	Sausage	1 medium sized sausage	20
13	Puddings	1 average helping of milk pudding	20
		1 average helping of steamed pudding	40
14	Fruit	1 banana	20
		1 apple	12
		1 orange	12
		1 average helping stewed fruit	18
		1 prune or date	5
15	Vegetables	2 dessertspoonfuls of cooked peas, lentils or baked beans	10
16	Milk	1 pint	30
17	Beer	1 pint	20

Raw sugar, jam and sweets were excluded from the diet. Sugar was, however, present in scones, cakes, etc.

CONVERSION TABLE FOR PROTEIN FOODS.

	Item	Quantity	Approximate Protein Content. Grammes
1	Meat	1 average helping	15
2	Fish	1 average helping	15
3	Egg	1	7
4	Cheese	1 oz.	7
5	Bacon	1 small helping	6
6	Milk	1 pint	20
7	Bread	1 slice national loaf	4

The protein contents of peas, beans and cakes were omitted from the calculations.

The quantity of protein consumed by diabetics on a free diet was greater than that consumed by the general population because of the extra rations of meat, cheese, bacon and milk received by diabetics.

ESTIMATION OF THE DIETARY FAT CONTENTS.

During the period of observation, diabetics received three times the fat ration allowed to the general population. The latter allowance was 30 grammes of butter or margarine daily, which, along with the fat obtained from rations of meat, fish, cheese, eggs and milk, provided a normal fat ration of approximately 60 grammes daily.

The diabetic on a free diet in many instances shared his extra ration with his family and the fat content of his diet was approximately 60 grammes. When he took two thirds of his ration, it was 90 grammes and, if he consumed all his ration, about 120 grammes.

APPENDIX.

Part II.

INSULIN SENSITIVITY TESTS.

Free Diet Group.

	Initials	Fasting Blood Sugar Mg.%	Lowest Blood Sugar recorded Mg.%	Absolute Fall in Mg.	% Fall of Blood Sugar	Age. Years	Average Daily Carbo-hydrate Intake. Grammes	24 hour Glyco-suria Grammes %
1	T.E.	350	229	121	35	37	277	5.2
2	D.P.	421	286	135	32	62	229	4.0
3	B.R.	321	189	132	41	16	459	5.1
4	L.H.	291	226	65	22	16	550	5.7
5	E.H.	298	149	149	50	49	242	4.4
6	J.McWm.	263	198	65	25	37	274	4.0
7	G.S.	313	201	112	36	53	218	2.0
8	M.Mc.	294	154	140	48	27	502	4.7
9	A.D.	277	112	165	60	33	289	3.1
10	R.Ga.	153	91	62	41	31	246	4.6
11	D.W.	230	164	66	29	31	289	3.1
12	J.J.	300	192	108	36	38	354	5.1
13	J.B.	262	150	112	43	29	440	4.5
14	J.S.	117	60	57	49	61	317	1.4
15	T.K.	360	237	123	34	37	264	3.2
16	D.C.	288	135	153	53	15	405	6.2
17	P.M.	191	36	155	81	31	371	1.1

INSULIN SENSITIVITY TESTS.

Control Group.

	Initials	Fasting Blood Sugar Mg.%	Lowest Blood Sugar recorded Mg.%	Absolute Fall in Mg.	% Fall of Blood Sugar	Age. Years	Average Daily Carbo-hydrate Intake. Grammes	24 hour Glyco-suria Grammes %
1	J.Mc.	260	103	157	60	34	175	1.2
2	A.P.	180	48	132	73	41	220	0.8
3	G.N.	293	170	123	42	44	250	3.6
4	H.S.	270	135	135	50	48	200	5.6
5	K.Mc.	245	149	96	39	35	175	2.4
6	D.G.	212	32	180	85	35	200	0
7	L.	163	50	113	69	68	130	0.6
8	S.	100	42	58	58	57	160	0
9	D.	101	40	61	60	37	175	0
10	M.	128	33	95	74	67	160	0

On Diet alone

BLOOD SUGAR CURVES.

	Initials	Date of Curve	Relation to Onset of Treatment	Fasting Blood Sugar Mg.%	Blood Sugar after 1 hour Mg.%	Blood Sugar after 2 hours Mg.%	Level after 2 hours compared with level after 1 hour Mg.
1	T.E.	24. 6.46	After	269	459	387	- 72
		28. 9.47	"	237	464	402	- 62
		12. 6.48	"	281	442	395	- 47
2	D.P.	27.11.45	After	227	288	390	+ 102
		21. 3.46	"	191	318	331	+ 13
		18.10.46	"	301	404	449	+ 45
		21. 2.47	"	315	425	436	+ 11
		14. 7.47	"	342	506	420	- 86
		4.12.47	"	294	342	328	- 86
		3. 6.48	"	402	504	494	- 10
3	R.Gr.	3.12.45	After	256	336	280	- 56
		1. 4.46	"	220	338	277	- 61
		15.10.46	"	263	361	293	- 68
		3. 3.47	"	302	421	341	- 80
		27. 7.47	"	206	384	292	- 92
		22. 5.48	"	229	412	341	- 71
4	B.R.	8. 1.46	Before	201	288	319	+ 31
		26.10.46	After	333	423	459	+ 36
		27. 2.47	"	331	376	257	- 19
		6. 5.48	"	247	310	436	+ 126

BLOOD SUGAR CURVES

	Initials	Date of Curve	Relation to Onset of Treatment	Fasting Blood Sugar Mg.%	Blood Sugar after 1 hour Mg.%	Blood Sugar after 2 hours Mg.%	Level after 2 hours compared with level after 1 hour Mg.
5	J.Di.	7.12.45	Before	264	495	452	- 43
		27. 3.46	After	165	400	272	- 128
		18. 2.47	"	126	380	331	- 49
		24. 7.47	"	119	375	392	+ 17
6	L.H.	28.12.45	After	81	341	264	- 77
		21. 3.46	"	67	197	203	+ 6
		23.10.46	"	300	424	433	+ 9
		20. 2.47	"	241	410	390	- 20
		23. 7.47	"	231	284	284	Same
		12. 5.48	"	312	417	358	- 59
7	R.McW.	10. 1.46	After	317	514	466	- 48
		16. 4.46	"	188	252	237	- 15
		27.10.46	"	173	455	405	- 50
		24. 2.47	"	274	436	392	- 44
		5. 6.48	"	173	340	318	- 22
8	J.McW.	22. 1.46	After	190	418	309	- 109
		9. 4.46	"	200	412	336	- 76
		27.10.46	"	177	414	426	+ 12
		30. 5.48	"	207	323	295	- 28

BLOOD SUGAR CURVES.

	Initials	Date of Curve	Relation to Onset of Treatment	Fasting Blood Sugar Mg.%	Blood Sugar after 1 hour Mg.%	Blood Sugar after 2 hours Mg.%	Level after 2 hours compared with level after 1 hour Mg.
9	E.H.	11. 2.46	After	178	363	399	+ 36
		4. 4.46	"	76	165	143	- 22
		17.10.46	"	130	285	280	- 5
		22. 7.47	"	312	432	379	- 53
		15. 5.48	"	367	531	534	+ 3
10	J.McWm.	4. 3.46	After	89	267	200	- 67
		1. 3.47	"	152	268	236	- 32
		25.11.47	"	110	280	222	- 58
		8. 6.48	"	201	380	325	- 55
11	H.S.	8. 3.46	After	251	440	326	- 114
		16. 4.46	"	243	485	381	- 104
		29.10.46	"	304	464	502	+ 38
		19. 2.47	"	209	404	380	- 24
		25. 5.48	"	330	408	365	- 43
12	R.M.	4. 4.46	Before	174	330	278	- 52
		23. 4.46	After	207	346	293	- 53
		20.10.46	"	198	340	306	- 34
		26. 2.47	"	126	228	198	- 30
		23. 6.47	"	136	311	300	- 11
		23. 5.48	"	109	199	264	+ 65

BLOOD SUGAR CURVES.

	Initials	Date of Curve	Relation to Onset of Treatment	Fasting Blood Sugar Mg.%	Blood Sugar after 1 hour Mg.%	Blood Sugar after 2 hours Mg.%	Level after 2 hours compared with level after 1 hour Mg.
13	J.L.	7. 5.46	After	246	481	637	+156
		22.10.46	"	385	589	558	- 31
		25. 2.47	"	212	467	445	- 22
		16. 7.47	"	180	370	471	+ 101
		14. 5.48	"	400	550	592	+ 42
14	G.S.	3. 1.47	After	404	572	595	+ 23
		20. 7.47	"	308	409	394	- 15
		27. 5.48	"	286	506	520	+ 14
15	J.F.	20. 4.47	After	189	337	253	- 84
		21. 7.47	"	119	250	209	- 41
		6. 6.48	"	173	375	298	- 77
16	A.C.	7. 2.47	Before	210	383	361	- 22
		29. 7.47	After	322	423	388	- 35
		11. 5.48	"	207	323	335	+ 12
17	J.J.	18. 4.47	After	294	496	396	- 100
		26. 7.47	"	212	409	343	- 66
		22.11.47	"	266	440	385	- 55
		19. 6.48	"	245	436	385	- 51
18	D.C.	11.10.47	Before	308	500	486	- 14
		26.11.47	After	249	430	369	- 61
		16. 6.48	"	319	413	440	+ 27

APPENDIX.

Part III.

FREE DIET GROUP.

(a) General Information.

	Initials	Sex	Age in 1949. Years	Family History of Diabetes	Occupation	Energy Grade I, II, or III.
1	T.E.	M	38	-	Miner	I
2	D.P.	M	63	-	Business Man	I
3	R.Gr.	M	41	+	Tradesman	II
4	B.R.	M	17	-	Tradesman	I
5	J.Di.	F	20	-	Housewife	I
6	L.H.	M	17	-	Schoolboy	I
7	A.A.	F	57	+	Housewife	I
8	R.McW.	M	24	+	Lorry Driver	I
9	J.McW.	M	24	+	Labourer	I
10	C.Wi.	M	23	-	Student	I
11	E.Hi.	F	50	-	Housewife	I
12	J.McWm.	M	38	-	Business Man	I
13	H.A.	M	43	-	Lorry Driver	-
14	H.S.	F	31	-	Housewife	I
15	J.Du.	M	33	-	Miner	I
16	R.M.	M	52	+	Tradesman	II
17	J.L.	M	12	-	Schoolboy	I
18	J.Ph.	F	28	-	Housewife	I
19	G.S.	M	53 (1948)	-	Unemployed	I
20	R.N.	M	28	-	Business Man	I

FREE DIET GROUP.

(a) General Information.

	Initials	Sex	Age in 1949. Years	Family History of Diabetes	Occupation	Energy Grade I, II, or III.
21	J.Mc.	M	27	-	Labourer	I
22	J.F.	M	35	-	Tradesman	I
23	A.S.	F	37	-	Housewife	I
24	A.C.	M	25	-	Tradesman	II
25	A.Mc.	F	23	+	Typist	II
26	M.Mc.	M	28	-	Tradesman	II
27	A.N.	M	57	+	Miner	I
28	E.Ber.	F	18	-	Typist	I
29	C.M.	F	34	-	Business Woman	II
30	A.D.	M	34	+	Labourer	III
31	T.M.	M	58	-	Unemployed	
32	J.McM.	M	31	-	Labourer	
33	V.S.	F	25	-	Housewife	II
34	R.Ga.	M	32	-	Labourer	I
35	D.W.	M	32	+	Miner	I
36	J.J.	M	39	-	Lorry Driver	I
37	J.B.	M	30	-	Business Man	I
38	F.Mc.	M	48	-	Labourer	III
39	J.Pa.	F	17	-	Shop Assistant	I
40	E.L.	F	61	-	Housewife	I

FREE DIET GROUP.

(a) General Information.

	Initials	Sex	Age in 1949. Years	Family History of Diabetes	Occupation	Energy Grade I, II, or III.
41	C.Wa.	F	17	-	Shop Assistant	I
42	J.M.	F	72	-	Housewife	I
43	J.S.	M	62	-	Labourer	I
44	H.F.	F	38	+	Housewife	I
45	T.K.	M	38	-	Miner	II
46	D.C.	M	16	+	Tradesman	I
47	P.M.	M	32	-	Tradesman	I
48	A.W.	M	28	-	Miner	I
49	W.T.	M	28	-	Business Man	I
50	H.G.	F	28	-	Housewife	I

	Initials	Sex	Age in 1949. Years	Family History of Diabetes	Occupation	Complication
51	E.He.	F	27	-	Housewife	Thyrotoxicosis
52	D.D.	M	55	-	Business Man	Thyrotoxicosis
53	F.P.	F	53	+	Housewife	Gastric Carcinoma
54	I.T.	F	12	+	Schoolgirl	Pulmonary Tuberculosis
55	E.Bel.	F	24	+	Housewife	Psychoneurosis

FREE DIET GROUP.

(b) Duration of diabetes and the methods of treatment employed.

	Initials	Duration of previous "Dietetic" Treatment	Duration of Free Diet Treatment	Duration of subsequent Restricted Diet Treatment	Total duration of Diabetes
		Years	Years	Years	Years
1	T.E.	-	$3\frac{5}{12}$	-	$3\frac{5}{12}$
2	D.P.	3	$3\frac{4}{12}$	-	$6\frac{4}{12}$
3	R.Gr.	-	$3\frac{4}{12}$	-	$3\frac{4}{12}$
4	B.R.	-	$2\frac{10}{12}$	$\frac{6}{12}$	$3\frac{4}{12}$
5	J.Di.	-	$3\frac{3}{12}$	-	$3\frac{3}{12}$
6	L.H.	-	$2\frac{9}{12}$	$\frac{6}{12}$	$3\frac{3}{12}$
7	A.A.	-	$\frac{11}{12}$	$2\frac{4}{12}$	$3\frac{3}{12}$
8	R.McW.	9	$3\frac{2}{12}$	-	$12\frac{2}{12}$
9	J.McW.	4	$3\frac{2}{12}$	-	$7\frac{2}{12}$
10	C.Wi.	-	$3\frac{1}{12}$	-	$3\frac{1}{12}$
11	E.Hi.	-	$3\frac{1}{12}$	-	$3\frac{1}{12}$
12	J.McWm.	$\frac{6}{12}$	$3\frac{1}{12}$	-	$3\frac{7}{12}$
13	H.A.	20	$3\frac{1}{12}$	-	$23\frac{1}{12}$
14	H.S.	-	3	-	3
15	J.Du.	$1\frac{2}{12}$	3	-	$4\frac{2}{12}$
16	R.M.	-	3	-	3
17	J.L.	2	$2\frac{6}{12}$	$\frac{5}{12}$	$4\frac{11}{12}$
18	J.Ph.	-	$2\frac{6}{12}$	-	$2\frac{6}{12}$
19	G.S.	10	$1\frac{10}{12}$	Died	$11\frac{10}{12}$
20	R.N.	4	$2\frac{2}{12}$	-	$6\frac{2}{12}$

FREE DIET GROUP.

(b) Duration of diabetes and the methods of treatment employed.

	Initials	Duration of previous "Dietetic" Treatment	Duration of Free Diet Treatment	Duration of subsequent Restricted Diet Treatment	Total duration of Diabetes
		Years	Years	Years	Years
21	J.Mc.	-	$2\frac{2}{12}$	-	$2\frac{2}{12}$
22	J.F.	-	$2\frac{1}{12}$	$\frac{1}{12}$	$2\frac{2}{12}$
23	A.S.	$2\frac{8}{12}$	$2\frac{1}{12}$	-	$4\frac{9}{12}$
24	A.C.	-	$2\frac{1}{12}$	-	$2\frac{1}{12}$
25	A.Mc.	1	$1\frac{2}{12}$	$\frac{10}{12}$	3
26	M.Mc.	$\frac{7}{12}$	2	-	$2\frac{7}{12}$
27	A.N.	$1\frac{10}{12}$	$1\frac{7}{12}$	$\frac{5}{12}$	$3\frac{10}{12}$
28	E.Ber.	$1\frac{6}{12}$	2	-	$3\frac{6}{12}$
29	C.M.	$6\frac{6}{12}$	2	-	$8\frac{6}{12}$
30	A.D.	6	2	-	8
31	T.M.	-	2	-	2
32	J.McM.	8	2	-	10
33	V.S.	20	2	-	22
34	R.Ga.	1	$1\frac{11}{12}$	-	$2\frac{11}{12}$
35	D.W.	$\frac{10}{12}$	$1\frac{11}{12}$	-	$2\frac{9}{12}$
36	J.J.	-	$1\frac{11}{12}$	-	$1\frac{11}{12}$
37	J.B.	$1\frac{4}{12}$	$1\frac{11}{12}$	-	$3\frac{3}{12}$
38	F.Mc.	$2\frac{3}{12}$	$1\frac{11}{12}$	-	$4\frac{2}{12}$
39	J.Pa.	-	1	$\frac{11}{12}$	$1\frac{11}{12}$
40	E.L.	$\frac{8}{12}$	$1\frac{9}{12}$	-	$2\frac{5}{12}$

FREE DIET GROUP.

(b) Duration of diabetes and the methods of treatment employed.

	Initials	Duration of previous "Dietetic" Treatment	Duration of Free Diet Treatment	Duration of subsequent Restricted Diet Treatment	Total duration of Diabetes
		Years	Years	Years	Years
41	C.Wa.	$\frac{5}{12}$	$1\frac{3}{12}$	$\frac{5}{12}$	$2\frac{1}{12}$
42	J.M.	-	$1\frac{7}{12}$	-	$1\frac{7}{12}$
43	J.S.	-	$1\frac{7}{12}$	-	$1\frac{7}{12}$
44	H.F.	4	$1\frac{5}{12}$	-	$5\frac{5}{12}$
45	T.K.	$2\frac{1}{12}$	$1\frac{5}{12}$	-	$3\frac{6}{12}$
46	D.C.	-	$\frac{6}{12}$	1	$1\frac{6}{12}$
47	P.M.	-	$1\frac{4}{12}$	-	$1\frac{4}{12}$
48	A.W.	-	$1\frac{1}{12}$	-	$1\frac{1}{12}$
49	W.T.	$\frac{7}{12}$	1	-	$1\frac{7}{12}$
50	H.G.	$3\frac{6}{12}$	$\frac{4}{12}$	$\frac{5}{12}$	$4\frac{3}{12}$

FREE DIET GROUP.

(c) Insulin dosage.

	Initials	Daily Number of Insulin Injections	Distribution of Insulin Dosage		Daily Total Dose of Insulin March 1949
			Morning	Evening	
			Units	Units	Units
1	T.E.	1	24 Z.P.I. + 12 Sol.	-	36
2	D.P.	1	16 Z.P.I. + 28 Sol.	-	44
3	R.Gr.	2	20 Globin	16 Globin	36
4	B.R.	2	32 Z.P.I. + 24 Sol.	20 Sol.	76
5	J.Di.	2	32 Z.P.I. + 12 Sol.	12 Sol.	56
6	L.H.	2	40 Z.P.I. + 28 Sol.	20 Sol.	88
7	A.A.	2	8 Sol.	8 Sol.	16
8	R.McW.	2	32 Z.P.I. + 16 Sol.	16 Sol.	64
9	J.McW.	2	24 Z.P.I. + 20 Sol.	20 Sol.	64
10	C.Wi.	2	28 Z.P.I. + 24 Sol.	24 Sol.	76
11	E.Hi.	1	12 Z.P.I. + 8 Sol.	-	20
12	J.McWm.	2	12 Z.P.I. + 12 Sol.	12 Sol.	36
13	H.A.	2	20 Z.P.I. + 20 Sol.	20 Sol.	60
14	H.S.	2	36 Z.P.I. + 12 Sol.	24 Sol.	72
15	J.Du.	2	40 Sol.	28 Sol.	68
16	R.M.	2	36 Globin	20 Sol.	56
17	J.L.	2	22 Globin	20 Globin	42
18	J.Ph.	1	16 Z.P.I. + 12 Sol.	-	28
19	G.S.	2	12 Z.P.I. + 12 Sol.	12 Sol.	36
20	R.N.	2	20 Sol.	20 Sol.	40

FREE DIET GROUP.

(c) Insulin Dosage.

	Initials	Daily Number of Insulin Injections	Distribution of Insulin Dosage		Daily Total Dose of Insulin March 1949
			Morning	Evening	
			Units	Units	Units
21	J.Mc.	1	20 Z.P.I. + 20 Sol.	-	40
22	J.F.	2	32 Z.P.I. + 32 Sol.	36 Sol.	100
23	A.S.	2	12 Z.P.I. + 12 Sol.	12 Sol.	36
24	A.C.	2	36 Z.P.I. + 12 Sol.	12 Sol.	60
25	A.Mc.	2	44 Z.P.I. + 20 Sol.	20 Sol.	84
26	M.Mc.	2	28 Z.P.I. + 12 Sol.	16 Sol.	56
27	A.N.	2	16 Z.P.I. + 12 Sol.	20 Sol.	48
28	E.Ber.	2	16 Z.P.I. + 16 Sol.	16 Sol.	48
29	C.M.	2	16 Z.P.I. + 28 Sol.	12 Sol.	56
30	A.D.	2	8 Z.P.I. + 6 Sol.	6 Sol.	20
31	T.M.	1	20 Globin	-	20
32	J.McM.	2	12 Z.P.I. + 12 Sol.	12 Sol.	36
33	V.S.	2	28 Sol.	16 Sol.	44
34	R.Ga.	2	32 Z.P.I. + 28 Sol.	20 Sol.	80
35	D.W.	2	12 Z.P.I. + 12 Sol.	12 Sol.	36
36	J.J.	2	12 Z.P.I. + 12 Sol.	12 Sol.	36
37	J.B.	2	16 Z.P.I. + 12 Sol.	12 Sol.	40
38	F.Mc.	2	24 Z.P.I. + 16 Sol.	24 Sol.	64
39	J.Pa.	2	36 Z.P.I. + 28 Sol.	28 Sol.	92
40	E.L.	1	12 Globin	-	12

FREE DIET GROUP.

(c) Insulin Dosage.

	Initials	Daily Number of Insulin Injections	Distribution of Insulin Dosage		Daily Total Dose of Insulin March 1949
			Morning	Evening	
			Units	Units	Units
41	C.Wa.	2	44 Globin	40 Globin	84
42	J.M.	1	22 Globin	-	22
43	J.S.	1	20 Z.P.I.	-	20
44	H.F.	2	14 Z.P.I. + 20 Sol.	14 Sol.	48
45	T.K.	1	30 Z.P.I.	-	30
46	D.C.	2	40 Z.P.I. + 20 Sol.	20 Sol.	80
47	P.M.	2	12 Z.P.I. + 8 Sol.	12 Sol.	32
48	A.W.	2	12 Sol.	12 Sol.	24
49	W.T.	2	24 Globin	20 Globin	44
50	H.G.	2	24 Z.P.I. + 12 Sol.	12 Sol.	48

FREE DIET GROUP.

(d) Diets.

	Initials	Average Daily Carbo-hydrate	Average Daily Fat	Average Daily Protein	Average Daily Calorie Intake	Maximum Variation of Carbo-hydrate Intake from Monday to Sunday	Maximum Variation of Carbo-hydrate Intake from Monday to Friday
		Grammes	Grammes	Grammes		Grammes	Grammes
1	T.E.	277	120	113	2640	121	40
2	D.P.	229	120	95	2376	86	43
3	R.Gr.	306	60	129	2280	96	85
4	B.R.	459	120	105	3336	173	101
5	J.Di.	-	-	-	-	-	-
6	L.H.	551	120	116	3748	141	80
7	A.A.	-	-	-	-	-	-
8	R.McW.	363	90	134	2798	59	39
9	J.McW.	506	90	111	3278	226	114
10	C.Wi.	454	90	133	3158	96	96
11	E.Hi.	242	60	93	1880	66	63
12	J.McWm.	274	90	99	2302	106	92
13	H.A.	-	-	-	-	-	-
14	H.S.	291	90	102	2382	80	56
15	J.Du.	316	60	111	2248	205	83
16	R.M.	309	120	104	2732	147	147
17	J.L.	267	90	88	2230	80	80
18	J.Ph.	264	60	74	1892	93	72
19	G.S.	218	120	90	2312	76	69
20	R.N.	-	-	-	-	-	-

FREE DIET GROUP.

(d) Diets.

	Initials	Average Daily Carbo-hydrate	Average Daily Fat	Average Daily Protein	Average Daily Calorie Intake	Maximum Variation of Carbo-hydrate Intake from Monday to Sunday	Maximum Variation of Carbo-hydrate Intake from Monday to Friday
		Grammes	Grammes	Grammes		Grammes	Grammes
21	J.Mc.	428	120	118	3264	306	86
22	J.F.	347	120	112	2916	125	125
23	A.S.	225	120	99	2376	75	75
24	A.C.	330	120	128	2912	65	30
25	A.Mc.	375	120	103	2992	46	40
26	M.Mc.	502	120	114	3544	103	103
27	A.N.	398	90	134	2938	101	90
28	E.Ber.	338	90	88	2514	58	42
29	C.M.	256	120	90	2464	98	93
30	A.D.	289	90	90	2326	124	120
31	T.M.	-	-	-	-	-	-
32	J.McM.	-	-	-	-	-	-
33	V.S.	250	90	109	2246	105	49
34	R.Ga.	246	120	90	2424	33	30
35	D.W.	289	120	94	2612	165	28
36	J.J.	354	90	125	2726	53	53
37	J.B.	440	90	136	3114	169	36
38	F.Mc.	-	-	-	-	-	-
39	J.Pa.	448	120	110	3312	115	70
40	E.L.	239	90	81	2090	69	52

FREE DIET GROUP.

(d) Diets.

	Initials	Average Daily Carbo-hydrate	Average Daily Fat	Average Daily Protein	Average Daily Calorie Intake	Maximum Variation of Carbo-hydrate Intake from Monday to Sunday	Maximum Variation of Carbo-hydrate Intake from Monday to Friday
		Grammes	Grammes	Grammes		Grammes	Grammes
41	C.Wa.	323	90	92	2470	198	103
42	J.M.	217	120	67	2216	47	47
43	J.S.	317	120	120	2828	40	40
44	H.F.	297	60	83	2060	125	100
45	T.K.	264	120	104	2552	93	58
46	D.C.	405	120	120	3180	40	30
47	P.M.	371	120	127	3072	106	82
48	A.W.	-	-	-	-	-	-
49	W.T.	-	-	-	-	-	-
50	H.G.	-	-	-	-	-	-

FREE DIET GROUP.

(e) Ophthalmoscopic Findings.

	Initials	Age in Years. 1949	Duration of Diabetes in Years	Retinitis present	Cataract present
1	T.E.	38	3	No	No
2	D.P.	63	6	No	No
3	R.Gr.	41	3	No	No
4	B.R.	17	3	No	No
5	J.Di.	20	3	No	No
6	L.H.	17	3	No	No
7	A.A.	-	-	-	-
8	R.McW.	24	12	Early R. eye	No
9	J.McW.	24	7	No	No
10	C.Wi.	23	3	No	No
11	E.Hi.	50	3	No	No
12	J.McWm.	38	3	No	No
13	H.A.	-	-	-	-
14	H.S.	31	3	No	No
15	J.Du.	33	4	No	No
16	R.M.	52	3	No	No
17	J.L.	12	4	No	No
18	J.Ph.	28	2	No	No
19	G.S.	-	-	-	-
20	R.N.	-	-	-	-

FREE DIET GROUP.

(e) Ophthalmoscopic Findings.

	Initials	Age in Years. 1949	Duration of Diabetes in Years	Retinitis present	Cataract present
21	J.Mc.	-	-	-	-
22	J.F.	35	2	No	No
23	A.S.	37	4	No	No
24	A.C.	25	2	No	No
25	A.Mc.	23	3	No	No
26	M.Mc.	28	2	No	No
27	A.N.	57	3	No	No
28	E.Ber.	18	3	No	No
29	C.M.	34	8	No	No
30	A.D.	34	8	Early R. eye	No
31	T.M.	-	-	-	-
32	J.McM.	-	-	-	-
33	V.S.	25	22	No	No
34	R.Ga.	32	2	No	No
35	D.W.	32	2	No	No
36	J.J.	39	1	No	No
37	J.B.	30	3	No	No
38	F.Mc.	48	4	No	No
39	J.Pa.	17	1	No	No
40	E.L.	61	2	Early both eyes	No

FREE DIET GROUP.

(e) Ophthalmoscopic Findings.

	Initials	Age in Years. 1949	Duration of Diabetes in Years	Retinitis present	Cataract present
41	C.Wa.	17	2	No	No
42	J.M.	72	1	No	Early both eyes
43	J.S.	62	1	No	No
44	H.F.	38	5	No	No
45	T.K.	38	3	No	No
46	D.C.	16	1	No	No
47	P.M.	32	1	No	No
48	A.W.	28	1	No	No
49	W.T.	28	1	Early both eyes	No
50	H.G.	28	4	No	No
51	E.He.	27	4	No	No
52	D.D.	55	10	No	No

FREE DIET GROUP.

(f) Weights, 1949.

	Initials	Height	Weight St:lb.	Correct Weight St:lb.	Average Weight St:lb.	Highest Weight St:lb.
1	T.E.	5'7½"	10: 5	11: 0	10:10	10:10
2	D.P.	5'7½"	10:12	11: 4	11: 4	13: 0
3	R.Gr.	5'7½"	10: 2	11: 2	10:11	10:11
4	B.R.	5'7"	8:10	9: 6	-	-
5	J.Di.	5'1½"	7:13	8: 5	-	-
6	L.H.	5'9"	9: 2	10: 2	-	-
7	A.A.	5'4½"	11: 9	10: 6	10: 8	12:10
8	R.McW.	5'7½"	11: 0	10: 6	-	-
9	J.McW.	5'7"	10: 4	10: 4	-	-
10	C.Wi.	6'1"	11:12	12: 0	10: 0	10: 0
11	E.Hi.	5'1"	9: 2	9: 9	9: 4	11: 0
12	J.McWm.	6'0½"	12:13	12:12	13:10	13:10
13	H.A.	-	-	-	-	-
14	H.S.	5'7½"	7: 9	10: 5	7:12	7:12
15	J.Du.	5'6"	10: 7	10: 6	10: 6	10: 6
16	R.M.	5'4½"	10: 4	10: 6	9:12	9:12
17	J.L.	4'10"	6: 0	6: 0	-	-
18	J.Ph.	5'3½"	10: 6	9: 2	10: 0	12: 8
19	G.S.	5'8½"	11: 8	11: 9	11: 7	11:10
20	R.N.	5'4½"	10: 1	9:11	10: 0	10: 0

FREE DIET GROUP.

(f) Weights, 1949.

	Initials	Height	Weight St:lb.	Correct Weight St:lb.	Average Weight St:lb.	Highest Weight St:lb.
21	J.Mc.	5'8½"	11: 6	10:13	10: 8	12: 0
22	J.F.	6'0"	10: 3	12: 8	9:10	9:10
23	A.S.	5'2½"	9:12	9: 6	10: 0	10: 0
24	A.C.	6'0½"	12: 7	12: 2	13: 0	14: 4
25	A.Mc.	5'2½"	8: 4	8:10	7:12	7:12
26	M.Mc	5'10½"	10:10	11: 8	10:12	10:12
27	A.N.	5'4½"	10: 4	10: 6	10: 4	10: 4
28	E.Ber.	5'2"	7: 4	8: 5	7: 0	7: 0
29	C.M.	5'9"	10: 5	11: 0	10: 2	10: 2
30	A.D.	5'6"	9: 8	10: 6	10: 3	10: 3
31	T.M.	-	-	-	-	-
32	J.McM.	-	-	-	-	-
33	V.S.	5'5½"	10: 2	9: 6	-	-
34	R.Ga.	5'9"	11: 8	11: 3	12: 1	12: 1
35	D.W.	5'9½"	10: 8	11: 6	10: 7	10: 7
36	J.J.	5'8½"	12: 0	11: 6	12: 2	12: 2
37	J.B.	5'6½"	10: 2	10: 7	11: 0	11: 7
38	F.Mc.	5'8½"	10: 6	11:10	10: 8	10: 8
39	J.Pa.	5'7"	8: 6	9: 7	-	-
40	E.L.	5'7"	8: 2	11: 2	7: 6	7: 6

FREE DIET GROUP.

(f) Weights, 1949.

	Initials	Height	Weight St:lb.	Correct Weight St:lb.	Average Weight St:lb.	Highest Weight St:lb.
41	C.Wa.	5'2½"	8:10	8: 6	-	-
42	J.M.	5'1½"	8: 5	9:11	10: 0	12: 0
43	J.S.	5'7½"	12:11	11: 5	12: 8	12: 8
44	H.F.	5'2"	9: 2	9: 4	9: 0	9: 0
45	T.K.	5'8½"	11: 7	11: 7	12: 0	13: 7
46	D.C.	5'6"	9: 1	9: 2	-	-
47	P.M.	6'0"	10: 7	12: 5	9:11	9:11
48	A.W.	5'6"	10:11	10: 3	11: 0	11: 0
49	W.T.	6'0"	10: 2	12: 2	11: 0	11: 0
50	H.G.	5'5"	9: 8	9: 7	10: 4	11: 0

FREE DIET GROUP.

(g) Weight Relations.

	Initials	Highest Weight to Correct Weight: Precursorily Obese	Highest Weight to Correct Weight: Precursorily Normal	Highest Weight to Correct Weight: Precursorily Thin	Weight to Correct Weight	Weight to Average Weight	Weight to Highest Weight	Average Weight to Correct Weight
		St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.
1	T.E.		- 0: 4		- 0: 9	- 0: 5	- 0: 5	- 0: 4
2	D.P.	+ 1:10			- 0: 6	- 0: 6	- 2: 2	Same
3	R.Gr.		- 0: 5		- 1: 0	- 0: 9	- 0: 9	- 0: 5
4	B.R.				- 0:10			
5	J.Di.				- 0: 6			
6	L.H.				- 1: 0			
7	A.A.	+ 2: 4			+ 1: 3	+ 1: 1	- 1: 1	+0: 2
8	R.McW.				+ 0: 8			
9	J.McW.				Same			
10	C.Wi.			- 2: 0	- 0: 2	+ 1:12	+ 1:12	- 2: 0
11	E.Hi.	+ 1: 5			- 0: 7	- 0: 2	- 1:12	- 0: 5
12	J.McWm.	+ 0:12			+ 0: 1	- 0:11	- 0:11	+0:12
13	H.A.							
14	H.S.			- 2: 7	- 2:10	- 0: 3	- 0: 3	- 2: 7
15	J.Du.		Same		+ 0: 1	+ 0: 1	+ 0: 1	Same
16	R.M.			- 0: 8	- 0: 2	+ 0: 6	+ 0: 6	- 0: 8
17	J.L.				Same			
18	J.Ph.	+ 3: 6			+ 1: 4	+ 0: 6	- 2: 2	+0:12
19	G.S.		+ 0: 1		- 0: 1	+ 0: 1	- 0: 2	- 0: 2
20	R.N.		+ 0: 3		+ 0: 4	+ 0: 1	+ 0: 1	+ 0: 3

FREE DIET GROUP.

(g) Weight Relations.

	Initials	Highest Weight to Correct Weight: Precursorily Obese	Highest Weight to Correct Weight: Precursorily Normal	Highest Weight to Correct Weight: Precursorily Thin	Weight to Correct Weight	Weight to Average Weight	Weight to Highest Weight	Average Weight to Correct Weight
		St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.
21	J.Mc.	+ 1: 1			+ 0: 7	+ 0:12	- 0: 8	- 0: 5
22	J.F.			- 2:12	- 2: 5	+ 0: 7	+ 0: 7	- 2:12
23	A.S.	+ 0: 8			+ 0: 6	- 0: 2	- 0: 2	+ 0: 8
24	A.C.	+ 2: 2			+ 0: 5	- 0: 7	- 1:11	+ 0:12
25	A.Mc.			- 0:12	- 0: 6	+ 0: 6	+ 0: 6	- 0:12
26	M.Mc.			- 0:10	- 0:12	- 0: 2	- 0: 2	- 0:10
27	A.N.		- 0: 2		- 0: 2	Same	Same	- 0: 2
28	E.Ber.				- 1: 1			
29	C.M.			- 0:12	- 0: 9	+ 0: 3	+ 0: 3	- 0:12
30	A.D.		- 0: 3		- 0: 3	Same	Same	- 0: 3
31	T.M.							
32	J.McM.							
33	V.S.				+ 0:10			
34	R.Ga.	+ 0:12			+ 0: 5	- 0: 7	- 0: 7	+ 0:12
35	D.W.			- 0:13	- 0:12	+ 0: 1	+ 0: 1	- 0:13
36	J.J.	+ 0:10			+ 0: 8	- 0: 2	- 0: 2	+ 0:10
37	J.B.	+ 1: 0			- 0: 5	- 0:12	- 1: 5	+ 0: 7
38	F.Mc.			- 1: 2	- 1: 4	- 0: 2	- 0: 2	- 1: 2
39	J.Pa.				- 1: 1			
40	E.L.			- 3:10	- 3: 0	+ 0:10	+ 0:10	- 3:10

FREE DIET GROUP.

(g) Weight Relations.

	Initials	Highest Weight to Correct Weight			Weight to Correct Weight	Weight to Average Weight	Weight to Highest Weight	Average Weight to Correct Weight
		Precursorily Obese	Precursorily Normal	Precursorily Thin				
		St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.
41	C.Wa.				+ 0: 4			
42	J.M.	+ 2: 3			- 1: 6	- 1: 9	- 3: 9	+ 0: 3
43	J.S.	+ 1: 3			+ 1: 6	+ 0: 3	+ 0: 3	+ 1: 3
44	H.F.		- 0: 4		- 0: 2	+ 0: 2	+ 0: 2	- 0: 4
45	T.K.	+ 2: 0			Same	- 0: 7	- 2: 0	+ 0: 7
46	D.C.				- 0: 1			
47	P.M.			- 2: 8	- 1:12	+ 0:10	+ 0:10	- 2: 8
48	A.W.	+ 0:11			+ 0: 8	- 0: 3	- 0: 3	+ 0:11
49	W.T.			- 1: 2	- 2: 0	- 0:12	- 0:12	- 1: 2
50	H.G.	+ 1: 7			+ 0: 1	- 0:10	- 1: 6	+ 0:11

FREE DIET GROUP.

(h) Urine Sugar Losses.

	Initials	Sunday or week-day Collection	% Glycosuria in 24 hour Sample	Volume of 24 hour Sample	Carbohydrate Loss in 24 hours	Corresponding Average Intake of Carbohydrate	Daily Utilisation of Carbohydrate	Carbohydrate Loss as % of Intake
				Ml.	Grammes	Grammes	Grammes	
1	T.E.	W	5.2	2140	111	358	247	31%
2	D.P.	S	4.0	2620	105	289	184	39%
3	R.Gr.	W	3.6	2600	93	329	236	28%
4	B.R.	-	-	-	-	-	-	-
5	J.Di.	-	-	-	-	-	-	-
6	L.H.	-	-	-	-	-	-	-
7	A.A.	-	-	-	-	-	-	-
8	R.McW.	S	5.4	2675	156	366	210	43%
9	J.McW.	W	3.4	2400	88	426	338	21%
10	C.Wi.	S	2.5	2212	61	455	394	13%
11	E.Hi.	W	4.4	1880	84	247	163	34%
12	J.McWm.	S	4.0	2780	111	267	156	42%
13	H.A.	-	-	-	-	-	-	-
14	H.S.	W	5.1	2180	112	293	181	38%
15	J.Du.	W	4.2	2500	103	362	259	28%
16	R.M.	S	4.6	2960	136	342	206	40%
17	J.L.	-	-	-	-	-	-	-
18	J.Ph.	W	4.0	2000	80	260	180	31%
19	G.S.	W	2.0	1980	41	196	155	21%
20	R.N.	-	-	-	-	-	-	-

FREE DIET GROUP.

(h) Urine Sugar Losses.

	Initials	Sunday or week-day Collection	% Glycosuria in 24 hour Sample	Volume of 24 hour Sample	Carbohydrate Loss in 24 hours	Corresponding Average Intake of Carbohydrate	Daily Utilisation of Carbohydrate	Carbohydrate Loss as % of Intake
				Ml.	Grammes	Grammes	Grammes	
21	J.Mc.	-	-	-	-	-	-	-
22	J.F.	-	-	-	-	-	-	-
23	A.S.	W	4.4	1775	79	249	170	32%
24	A.C.	W	5.8	1960	116	375	259	31%
25	A.Mc.	-	-	-	-	-	-	-
26	M.Mc.	W	4.7	2250	106	461	355	23%
27	A.N.	-	-	-	-	-	-	-
28	E.Ber.	S	5.5	1475	81	239	158	34%
29	C.M.	W	5.6	1475	84	254	171	33%
30	A.D.	S	3.1	1200	40	258	218	16%
31	T.M.	-	-	-	-	-	-	-
32	J.McM.	-	-	-	-	-	-	-
33	V.S.	W	4.6	1950	92	243	151	38%
34	R.Ga.	W	4.6	1700	80	234	154	34%
35	D.W.	W	3.1	2175	67	298	231	22%
36	J.J.	W	5.1	2925	149	346	197	43%
37	J.B.	S	4.5	2300	102	409	307	25%
38	F.Mc.	-	-	-	-	-	-	-
39	J.Pa.	-	-	-	-	-	-	-
40	E.L.	W	3.1	2133	66	229	163	29%

FREE DIET GROUP.

(h) Urine Sugar Losses.

	Initials	Sunday or week-day Collection	% Glycosuria in 24 hour Sample	Volume of 24 hour Sample	Carbohydrate Loss in 24 hours	Corresponding Average Intake of Carbohydrate	Daily Utilisation of Carbohydrate	Carbohydrate Loss as % of Intake
				Ml.	Grammes	Grammes	Grammes	
41	C.Wa.	-	-	-	-	-	-	-
42	J.M.	W	3.2	2460	80	219	139	36%
43	J.S.	W	1.4	2300	33	318	285	10%
44	H.F.	W	4.1	2000	89	260	171	34%
45	T.K.	W	3.2	1740	57	292	235	19%
46	D.C.	-	-	-	-	-	-	-
47	P.M.	W	1.1	1725	19	370	351	5%
48	A.W.	-	-	-	-	-	-	-
49	W.T.	S	3.4	1475	50	255	205	19%
50	H.G.	-	-	-	-	-	-	-

FREE DIET GROUP.

(i) Blood Sugars.

	Initials	Fasting Sugar	Lowest Sugar	Highest Sugar	Fluctuation
		Mg.%	Mg.%	Mg.%	Mg.%
1	T.E.	-	-	-	-
2	D.P.	397	370	397	27
3	R.Gr.	-	-	-	-
4	B.R.	-	-	-	-
5	J.Di.	-	-	-	-
6	L.H.	-	-	-	-
7	A.A.	-	-	-	-
8	R.McW.	-	-	-	-
9	J.McW.	-	-	-	-
10	C.Wi.	56	56	198	142
11	E.Hi.	266	260	294	34
12	J.McWm.	178	178	288	110
13	H.A.	-	-	-	-
14	H.S.	325	282	380	98
15	J.Du.	301	119	301	182
16	R.M.	126	95	202	107
17	J.L.	-	-	-	-
18	J.Ph.	-	-	-	-
19	G.S.	303	63	303	240
20	R.N.	-	-	-	-

FREE DIET GROUP.

(i) Blood Sugars.

	Initials	Fasting Sugar	Lowest Sugar	Highest Sugar	Fluctuation
		Mg.%	Mg.%	Mg.%	Mg.%
21	J.Mc.	-	-	-	-
22	J.F.	-	-	-	-
23	A.S.	345	345	495	150
24	A.C.	198	77	308	231
25	A.Mc	-	-	-	-
26	M.Mc.	64	64	400	336
27	A.N.	-	-	-	-
28	E.Ber.	167	64	370	306
29	C.M.	198	198	232	34
30	A.D.	180	100	218	118
31	T.M.	-	-	-	-
32	J.McM.	-	-	-	-
33	V.S.	385	185	385	200
34	R.Ga.	64	64	162	98
35	D.W.	-	-	-	-
36	J.J.	120	120	232	112
37	J.B.	123	104	271	167
38	F.Mc.	-	-	-	-
39	J.Pa.	-	-	-	-
40	E.L.	230	230	337	107

FREE DIET GROUP.

(i) Blood Sugars.

	Initials	Fasting Sugar	Lowest Sugar	Highest Sugar	Fluctuation
		Mg.%	Mg.%	Mg.%	Mg.%
41	C.Wa.	-	-	-	-
42	J.M.	241	201	326	125
43	J.S.	109	109	221	112
44	H.F.	78	78	291	213
45	T.K.	109	109	236	127
46	D.C.	-	-	-	-
47	P.M.	123	112	123	11
48	A.W.	-	-	-	-
49	W.T.	280	77	413	336
50	H.G.	-	-	-	-

FREE DIET GROUP.

(j) Comparative Insulin Dosage.

	Initials	Dose of Insulin when stabilised on Free Diet Units	Dose of Insulin in March 1949 Units	Dose of Insulin when changed to Strict Diets Units	Relationship of recent dose to initial dose Units	% Relationship	Assessment = Higher dose Same dose Lower dose
1	T.E.	24	36		+ 12	+ 50%	Higher
2	D.P.	44	44		Same	Same	Same
3	R.Gr.	40	36		- 4	- 10	Same
4	B.R.	10		76	+ 66	+ 660	Higher
5	J.Di.	40	56		+ 16	+ 40	Higher
6	L.H.	36		88	+ 52	+ 144	Higher
7	A.A.	16		16	Same	Same	Same
8	R.McW.	68	64		- 4	- 6	Same
9	J.McW.	68	64		- 4	- 6	Same
10	C.Wi.	48	76		+ 28	+ 80	Higher
11	E.Hi.	10	20		+ 10	+ 100	Higher
12	J.McWm.	30	36		+ 6	+ 20	Higher
13	H.A.	-	-				-
14	H.S.	48	72		+ 24	+ 50	Higher
15	J.Du.	72	68		- 4	- 5	Same
16	R.M.	22	56		+ 34	+ 155	Higher
17	J.L.	28		42	+ 14	+ 50	Higher
18	J.Ph.	30	28		- 2	- 7	Same
19	G.S.	52	36		- 16	- 31	Lower
20	R.N.	48	40		- 8	- 17	Same

FREE DIET GROUP.

(j) Comparative Insulin Dosage.

	Initials	Dose of Insulin when stabilised on Free Diet Units	Dose of Insulin in March 1949 Units	Dose of Insulin when changed to Strict Diets Units	Relationship of recent dose to initial dose Units	% Relationship	Assessment = Higher dose Same dose Lower dose
21	J.Mc.	42	40		- 2	- 5	Same
22	J.F.	72		100	+ 28	+ 39	Higher
23	A.S.	44	36		- 8	- 18	Same
24	A.C.	60	60		Same	Same	Same
25	A.Mc.	54		84	+ 30	+ 56	Higher
26	M.Mc.	56	56		Same	Same	Same
27	A.N.	28		48	+ 20	+ 71	Higher
28	E.Ber.	48	48		Same	Same	Same
29	C.M.	58	56		- 2	- 3	Same
30	A.D.	30	20		- 10	- 33	Lower
31	T.M.	-	-				-
32	J.McM.	-	-				-
33	V.S.	44	44		Same	Same	Same
34	R.Ga.	48	72		+ 24	+ 50	Higher
35	D.W.	44	36		- 8	- 18	Same
36	J.J.	38	36		- 2	- 5	Same
37	J.B.	40	40		Same	Same	Same
38	F.Mc.	50	64		+ 14	+ 28	Higher
39	J.Pa.	44		92	+ 48	+ 109	Higher
40	E.L.	12	12		Same	Same	Same

FREE DIET GROUP.

(j) Comparative Insulin Dosage.

	Initials	Dose of Insulin when stabilised on Free Diet Units	Dose of Insulin in March 1949 Units	Dose of Insulin when changed to Strict Diets Units	Relationship of recent dose to initial dose Units	% Relationship	Assessment = Higher dose Same dose Lower dose
41	C.Wa.	84		84	Same	Same	Same
42	J.M.	16	22		+ 6	+ 38	Higher
43	J.S.	32	20		- 12	- 38	Lower
44	H.F.	48	48		Same	Same	Same
45	T.K.	30	30		Same	Same	Same
46	D.C.	52		80	+ 28	+ 54	Higher
47	P.M.	36	32		- 4	- 11	Same
48	A.W.	24	24		Same	Same	Same
49	W.T.	56	44		- 12	- 21	Lower
50	H.G.	48		48	Same	Same	Same

CONTROL GROUP.

(a) General Information.

	Initials	Sex	Age in 1949. Years	Family History of Diabetes	Occupation	Energy Grade I, II, or III.
1	E.J.	F	39	+	Housewife	III
2	S.B.	F	58	-	Housewife	III
3	M.Cr.	F	51	-	Housewife	I
4	J.Be.	F	54	+	Typist	I
5	E.R.	F	60	+	Housewife	III
6	M.H.	F	46	-	School Teacher	I
7	E.G.	F	62	-	Housewife	III
8	S.R.	M	18	-	Student	I
9	W.D.	M	18	-	Chemist	I
10	M.R.	F	49	-	Housewife	III
11	M.F.	F	67	-	Housewife	III
12	E.U.	F	46	-	Housewife	I
13	E.W.	F	55	-	Housewife	III
14	M.Ce.	F	19	+	Shop Assistant	I
15	E.Cam.	F	56	+	Shop Assistant	III
16	J.L.	M	48	-	Clerk	I
17	T.H.	M	39	-	Butcher	II
18	C.C.	F	20	-	Housewife	I
19	E.Cai.	F	32	+	Housewife	III
20	B.S.	F	64	-	Housewife	III

CONTROL GROUP.

(a) General Information.

	Initials	Sex	Age in 1949. Years	Family History of Diabetes	Occupation	Energy Grade I, II, or III.
21	J.Br.	M	48	-	Business Man	III
22	A.W.	M	23	-	Chemist	I
23	A.C.	F	49	+	Housewife	III
24	M.M.	F	33	+	Housewife	III
25	P.C.	M	49	-	Tram Conductor	I
26	U.C.	F	23	-	Typist	I
27	A.A.	M	26	-	Grocer	II
28	E.S.	F	45	-	Cashier	I
29	C.T.	F	29	-	Housewife	I
30	B.Mc.	F	59	+	Housewife	I
31	A.S.	M	13	-	Schoolboy	II
32	P.H.	M	16	+	Labourer	II
33	O.B.	M	13	-	Schoolboy	I
34	L.M.	F	42	-	Housewife	III
35	J.Mc.	M	35	-	Cashier	II
36	A.P.	M	42	-	Business Man	I
37	G.N.	M	45	-	Business Man	II
38	H.S.	M	49	-	Business Man	III
39	K.Mc.	M	36	-	Painter	I
40	D.G.	M	36	-	Chartered Accountant	III

CONTROL GROUP.

(b) Duration of Diabetes and Insulin Dosage.

	Initials	Total Duration of Diabetes	Daily Number of Insulin Injections	Distribution of Insulin Dosage		Daily Total Dose of Insulin March 1949
				Morning	Evening	
		Years		Units	Units	Units
1	E.J.	$1\frac{9}{12}$	1	14 Z.P.I.	-	14
2	S.B.	$\frac{11}{12}$	1	22 Z.P.I.	-	22
3	M.Cr.	$2\frac{7}{12}$	1	30 Z.P.I.	-	30
4	J.Be.	$1\frac{3}{12}$	1	38 G.	-	38
5	E.R.	13	1	12 Z.P.I.	-	12
6	M.H.	$4\frac{6}{12}$	1	20 G.	-	20
7	E.G.	$\frac{5}{12}$	1	20 Z.P.I.	-	20
8	S.R.	2	1	28 Z.P.I. + 14 Sol.	-	42
9	W.D.	1	1	36 Z.P.I.	-	36
10	M.R.	$7\frac{10}{12}$	2	28 Sol.	26 Sol.	54
11	M.F.	$\frac{7}{12}$	1	24 Z.P.I. + 10 Sol.	-	34
12	E.U.	$\frac{8}{12}$	1	10 Z.P.I.	-	10
13	E.W.	$10\frac{2}{12}$	2	22 Sol.	16 Sol.	38
14	M.Ce.	$3\frac{6}{12}$	2	26 Sol.	30 Sol.	56
15	E.Cam.	$\frac{11}{12}$	1	20 G.	-	20
16	J.L.	$16\frac{6}{12}$	2	32	24	56
17	T.H.	$7\frac{9}{12}$	1	34 Z.P.I. + 18 Sol.	-	52
18	C.C.	$\frac{10}{12}$	1	32 Z.P.I. + 16 Sol.	-	48
19	E.Cai.	$6\frac{3}{12}$	2	22 Sol.	22 Sol.	44
20	B.S.	9	1	10 G.	-	10

CONTROL GROUP.

(b) Duration of Diabetes and Insulin Dosage.

	Initials	Total Duration of Diabetes	Daily Number of Insulin Injections	Distribution of Insulin Dosage		Daily Total Dose of Insulin March 1949
				Morning	Evening	
		Years		Units	Units	Units
21	J.Br.	$7\frac{3}{12}$	2	22 Sol.	22 Sol.	44
22	A.W.	2	1	40 Z.P.I.	-	40
23	A.C.	$\frac{10}{12}$	1	18 Z.P.I.	-	18
24	M.M.	$2\frac{9}{12}$	1	30 Z.P.I.	-	30
25	P.C.	1	1	8 Z.P.I.	-	8
26	U.C.	2	1	24 Z.P.I.	-	24
27	A.A.	$2\frac{5}{12}$	1	30 Z.P.I.	-	30
28	E.S.	$2\frac{10}{12}$	1	20 Z.P.I.	-	20
29	C.T.	7	1	32 Z.P.I. + 10 Sol.	-	42
30	B.Mc.	$3\frac{2}{12}$	1	20 G.	-	20
31	A.S.	$1\frac{11}{12}$	1	40 Z.P.I. + 20 Sol.	-	60
32	P.H.	$3\frac{1}{12}$	2	40 Z.P.I. + 20 Sol.	20 Sol.	80
33	O.B.	$2\frac{4}{12}$	1	28 Z.P.I. + 14 Sol.	-	42
34	L.M.	9	1	32 Z.P.I.	-	32
35	J.Mc.	$1\frac{11}{12}$	1	14 Z.P.I.	-	14
36	A.P.	$2\frac{5}{12}$	1	12 Z.P.I.	-	12
37	G.N.	$1\frac{4}{12}$	2	20 Z.P.I. + 14 Sol.	14 Sol.	48
38	H.S.	$4\frac{4}{12}$	1	26 G.	-	26
39	K.Mc.	$2\frac{10}{12}$	1	30 G.	-	30
40	D.G.	2	1	22 Z.P.I.	-	22

CONTROL GROUP.

(c) Diets.

	Initials	Prescribed Daily Carbohydrate	Prescribed Daily Calorie Intake	Actual Daily Carbohydrate Intake	Difference between Actual Diet and Prescribed Diet
		Grammes		Grammes	Grammes
1	E.J.	145	2200	145	0
2	S.B.	145	2000	170	+ 25
3	M.Cr.	175	2400	190	+ 15
4	J.Be.	160	2200	160	0
5	E.R.	160	2000	145	- 15
6	M.H.	145	2000	145	0
7	E.G.	160	2000	160	0
8	S.R.	160	2400	160	0
9	W.D.	200	2240	220	+ 20
10	M.R.	160	1800	185	+ 25
11	M.F.	160	2200	185	+ 25
12	E.U.	145	2000	130	- 15
13	E.W.	175	2400	175	0
14	M.Ce.	160	2150	160	0
15	E.Cam.	180	2400	193	+ 13
16	J.L.	200	2700	160	- 40
17	T.H.	175	2450	175	0
18	C.C.	175	2100	175	0
19	E.Cai.	175	2400	185	+ 10
20	B.S.	130	1800	140	+ 10

CONTROL GROUP.

(c) Diets.

	Initials	Prescribed Daily Carbohydrate	Prescribed Daily Calorie Intake	Actual Daily Carbohydrate Intake	Difference between Actual Diet and Prescribed Diet
		Grammes		Grammes	Grammes
21	J.Br.	210	2400	230	+ 20
22	A.W.	190	2700	175	- 15
23	A.C.	175	2220	175	0
24	M.M.	175	2600	175	0
25	P.C.	145	2000	234	+ 89
26	U.C.	190	2700	190	0
27	A.A.	215	3000	217	+ 2
28	E.S.	145	2300	186	+ 41
29	C.T.	200	2420	207	+ 7
30	B.Mc.	160	1500	226	+ 66
31	A.S.	175	2300	184	+ 9
32	P.H.	200	2560	200	0
33	O.B.	175	2400	175	0
34	L.M.	160	2200	160	0
35	J.Mc.	175	2500	175	0
36	A.P.	175	2500	220	+ 45
37	G.N.	175	2500	250	+ 75
38	H.S.	200	2400	200	0
39	K.Mc.	175	2700	175	0
40	D.G.	200	2600	200	0

The daily protein intakes of the Control Group lie between 80-110 grammes.
The daily fat intakes, between 100-160 grammes.

CONTROL GROUP.

(d) Weights, 1949.

	Initials	Height	Weight St:lb.	Correct Weight St:lb.	Average Weight St:lb.	Highest Weight St:lb.
1	E.J.	5'3"	8:13	9: 9	9: 7	9: 7
2	S.B.	5'1"	9: 7	9: 9	11: 8	12: 8
3	M.Cr.	5'6"	9: 4	10:12	10: 0	11: 0
4	J.Be.	5'2½"	9: 2	10: 0	9: 8	9: 8
5	E.R.	5'2½"	10: 2	10: 0	10: 4	12: 4
6	M.H.	5'2"	9: 4	9:10	9: 7	9: 7
7	E.G.	4'11½"	9: 8	9: 6	10: 0	11: 0
8	S.R.	6'2"	12: 1	12: 0	-	-
9	W.D.	5'11"	10: 0	10:13	-	-
10	M.R.	5'5½"	11: 4	10:10	11:12	11:12
11	M.F.	5'4"	6:13	10: 4	8: 7	8: 7
12	E.U.	5'4"	11: 2	10: 1	12: 4	12: 4
13	E.W.	5'7½"	11: 1	11: 4	10: 6	13: 4
14	M.Ce.	5'1"	9: 7	8: 3	-	-
15	E.Cam.	5'4½"	9: 2	10: 6	9: 7	9: 7
16	J.L.	5'11½"	12: 6	12:11	14: 0	16: 0
17	T.H.	5'7½"	11: 1	11: 1	10:10	10:10
18	C.C.	5'6½"	10: 0	9: 7	11: 0	11: 0
19	E.Cai.	5'4½"	8: 3	9: 8	9: 0	9:12
20	B.S.	5'4"	10: 6	10: 4	9: 0	14: 0

CONTROL GROUP.

(d) Weights, 1949.

	Initials	Height	Weight St:lb.	Correct Weight St:lb.	Average Weight St:lb.	Highest Weight St:lb.
21	J.Br.	5'7"	10: 3	11: 2	10: 4	10: 4
22	A.W.	5'8"	10: 1	10: 7	11: 0	11: 0
23	A.C.	5'6½"	9: 3	11: 0	10: 7	11: 7
24	M.M.	5'6½"	7:12	10: 4	8:12	9: 4
25	P.C.	5'7"	12: 5	11: 2	12: 8	13: 4
26	U.C.	5'7½"	9: 4	10: 0	9:12	9:12
27	A.A.	5'7½"	11: 2	10: 7	11: 1	12: 0
28	E.S.	5'5"	8: 8	10: 5	10: 0	10: 4
29	C.T.	5'3½"	9: 9	9: 2	10: 2	10: 2
30	B.Mc.	5'4"	10:13	10: 4	11: 0	11: 8
31	A.S.	4'9"	5: 6	5:10	-	-
32	P.H.	5'8"	9: 2	9:10	-	-
33	O.B.	4'11"	6: 5	6: 2	-	-
34	L.M.	5'5½"	8: 5	10: 4	9:10	9:10
35	J.Mc.	5'4"	8:10	9:12	10:10	10:10
36	A.P.	5'7"	11: 4	11: 0	12: 4	12: 4
37	G.N.	5'11½"	11: 9	12:11	12: 4	12: 4
38	H.S.	5'7½"	9: 1	11: 4	10: 0	10: 0
39	K.Mc.	5'7½"	10:12	11: 0	10: 5	10: 5
40	D.G.	5'8"	10: 3	11: 3	11: 7	12: 8

CONTROL GROUP.

(e) Weight Relations.

	Initials	Highest Weight to Correct Weight			Weight to Correct Weight	Weight to Average Weight	Weight to Highest Weight	Average Weight to Correct Weight
		Precursorily Obese	Precursorily Normal	Precursorily Thin				
		St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.
1	E.J.		- 0: 2		- 0:10	- 0: 8	- 0: 8	- 0: 2
2	S.B.	+ 2:13			- 0: 2	- 2: 1	- 3: 1	+ 1:13
3	M.Cr.		+ 0: 2		- 1: 8	- 0:10	- 1:10	- 0:12
4	J.Be.		- 0: 6		- 0:12	- 0: 6	- 0: 6	- 0: 6
5	E.R.	+ 2: 4			+ 0: 2	- 0: 2	- 2: 2	+ 0: 4
6	M.H.		- 0: 3		- 0: 6	- 0: 3	- 0: 3	- 0: 3
7	E.G.	+ 1: 8			+ 0: 2	- 0: 6	- 1: 6	+ 0: 8
8	S.R.				+ 0: 1			
9	W.D.				- 0:13			
10	M.R.	+ 1: 2			+ 0: 8	- 0: 8	- 0: 8	+ 1: 2
11	M.F.			- 1:11	- 3: 5	- 1: 8	- 1: 8	- 1:11
12	E.U.	+ 2: 3			+ 1: 1	- 1: 2	- 1: 2	+ 2: 3
13	E.W.	+ 2: 0			- 0: 3	+ 0: 9	- 2: 3	- 0:12
14	M.Ce.				+ 1: 4			
15	E.Cam.			- 0:13	- 1: 4	- 0: 5	- 0: 5	- 0:13
16	J.L.	+ 3: 3			- 0: 5	- 1: 8	- 3: 8	+ 1: 3
17	T.H.		- 0: 5		Same	+ 0: 5	+ 0: 5	- 0: 5
18	C.C.	+ 1: 7			+ 0: 7	- 1: 0	- 1: 0	+ 1: 7
19	E.Cai.		+ 0: 4		- 1: 5	- 0:11	- 1: 9	- 0: 8
20	B.S.	+ 3:10			+ 0: 2	+ 1: 6	- 3: 8	- 1: 4

CONTROL GROUP.

(e) Weight Relations.

	Initials	Highest Weight to Correct Weight			Weight to Correct Weight	Weight to Average Weight	Weight to Highest Weight	Average Weight to Correct Weight
		Precursorily Obese	Precursorily Normal	Precursorily Thin				
		St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.	St:lb.
21	J.Br.			- 0:12	- 0:13	- 0: 1	- 0: 1	- 0:12
22	A.W.		+ 0: 7		- 0: 6	- 0:13	- 0:13	+ 0: 7
23	A.C.		+ 0: 7		- 1:11	- 1: 4	- 2: 4	- 0: 7
24	M.M.			- 1: 0	- 2: 6	- 1: 0	- 1: 6	- 1: 6
25	P.C.	+ 2: 2			+ 1: 3	- 0: 3	- 0:13	+ 1: 6
26	U.C.		- 0: 2		- 0:10	- 0: 8	- 0: 8	- 0: 2
27	A.A.	+ 1: 7			+ 0: 9	+ 0: 1	- 0:12	+ 0: 8
28	E.S.		- 0: 1		- 1:11	- 1: 6	- 1:10	- 0: 5
29	C.T.	+ 1: 0			+ 0: 7	- 0: 7	- 0: 7	+ 1: 0
30	B.Mc.	+ 1: 4			+ 0: 9	- 0: 1	- 0: 9	+ 0:10
31	A.S.				- 0: 4			
32	P.H.				- 0: 8			
33	O.B.				+ 0: 3			
34	L.M.			- 0: 8	- 1:13	- 1: 5	- 1: 5	- 0: 8
35	J.Mc.	+ 0:12			- 1: 2	- 2: 0	- 2: 0	+ 0:12
36	A.P.	+ 1: 4			+ 0: 4	- 1: 0	- 1: 0	+ 1: 4
37	G.N.		- 0: 7		- 1: 2	- 0: 9	- 0: 9	- 0: 7
38	H.S.			- 1: 4	- 2: 3	- 0:13	- 0:13	- 1: 4
39	K.Mc.			- 0: 9	- 0: 2	+ 0: 7	+ 0: 7	- 0: 9
40	D.G.	+ 1: 5			- 1: 0	- 1: 4	- 2: 5	+ 0: 4

CONTROL GROUP.

(f) Urine Sugar Losses.

	Initials	% Glycosuria in 24 hour Sample	Volume of 24 hour Sample	Carbohydrate Loss in 24 hours	Corresponding Intake of Carbohydrate	Daily Utilisation of Carbohydrate	Carbohydrate Loss as % of Intake
			Ml.	Grammes	Grammes	Grammes	
1	E.J.	0	1800	0	145	145	0
2	S.B.	2.2	2400	50	170	120	29
3	M.Cr.	3.4	2100	71	190	119	37
4	J.Be.	1.0	1800	18	160	142	11
5	E.R.	0	2400	0	145	145	0
6	M.H.	0	2400	0	145	145	0
7	E.G.	0	2000	0	160	160	0
8	S.R.	2.0	1500	30	160	130	19
9	W.D.	1.4	2000	28	220	192	13
10	M.R.	2.0	1500	30	185	155	16
11	M.F.	5.0	2700	135	185	50	73
12	E.U.	0	1600	0	130	130	0
13	E.W.	1.4	2000	28	175	147	16
14	M.Ce.	2.0	2000	40	160	120	25
15	E.Cam.	1.4	1800	25	193	168	13
16	J.L.	2.8	1800	50	160	110	31
17	T.H.	3.4	3000	102	175	73	58
18	C.C.	3.0	1500	45	175	130	26
19	E.Cai.	2.8	1800	50	185	135	27
20	B.S.	4.4	2400	106	140	34	76

CONTROL GROUP.

(f) Urine Sugar Losses.

	Initials	% Glycosuria in 24 hour Sample	Volume of 24 hour Sample	Carbohydrate Loss in 24 hours	Corresponding Intake of Carbohydrate	Daily Utilisation of Carbohydrate	Carbohydrate Loss as % of Intake
			Ml.	Grammes	Grammes	Grammes	
21	J.Br.	2.4	2800	67	230	163	29
22	A.W.	1.0	3000	30	175	145	17
23	A.C.	0.8	2400	19	175	156	11
24	M.M.	0	2100	0	175	175	0
25	P.C.	1.0	1800	18	234	216	8
26	U.C.	3.6	1500	54	190	136	28
27	A.A.	2.0	2400	48	217	169	22
28	E.S.	2.6	1800	47	186	139	25
29	C.T.	1.8	2100	38	207	169	18
30	B.Mc.	0.4	2490	10	226	216	4
31	A.S.	1.6	1800	28	184	156	15
32	P.H.	3.6	2400	86	200	114	43
33	O.B.	2.0	2000	40	175	135	23
34	L.M.	1.0	1500	15	160	145	9
35	J.Mc.	1.2	1800	21	175	154	12
36	A.P.	0.8	2000	16	220	204	7
37	G.N.	3.6	3100	110	250	140	44
38	H.S.	5.6	2400	134	200	66	67
39	K.Mc.	2.4	1800	43	175	132	25
40	D.G.	0	2000	0	200	200	0

CONTROL GROUP.

(g) Blood Sugar.

	Initials	Lowest Sugar	Highest Sugar	Fluctuation
		Mg.%	Mg.%	Mg.%
1	E.J.	76	177	101
2	S.B.	251	306	55
3	M.Cr.	257	265	8
4	J.Be.	176	211	35
5	E.R.	176	191	15
6	M.H.	107	135	28
7	E.G.	187	206	19
8	S.R.	71	221	150
9	W.D.	157	219	62
10	M.R.	50	242	192
11	M.F.	328	389	61
12	E.U.	80	105	25
13	E.W.	89	171	82
14	M.Ce.	158	215	57
15	E.Cam.	96	217	121
16	J.L.	-	-	-
17	T.H.	-	-	-
18	C.C.	112	126	14
19	E.Cai.	-	-	-
20	B.S.	236	395	159

CONTROL GROUP.

(g) Blood Sugar.

	Initials	Lowest Sugar	Highest Sugar	Fluctuation
		Mg.%	Mg.%	Mg.%
21	J.Br.	117	131	14
22	A.W.	135	217	82
23	A.C.	253	265	12
24	M.M.	-	-	-
25	P.C.	164	166	2
26	U.C.	232	395	163
27	A.A.	149	217	68
28	E.S.	191	236	45
29	C.T.	202	460	258
30	B.Mc.	84	196	112
31	A.S.	50	316	266
32	P.H.	178	318	140
33	O.B.	82	220	138
34	L.M.	142	242	100
35	J.Mc.	173	207	34
36	A.P.	77	102	25
37	G.N.	272	317	45
38	H.S.	-	-	-
39	K.Mc.	60	112	52
40	D.G.	-	-	-